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PROCTOLOGY

Papers Read at the June 1947
Meeting of
The American Proctologic Society

February 1948

VOLUME LXXV · NUMBER 2

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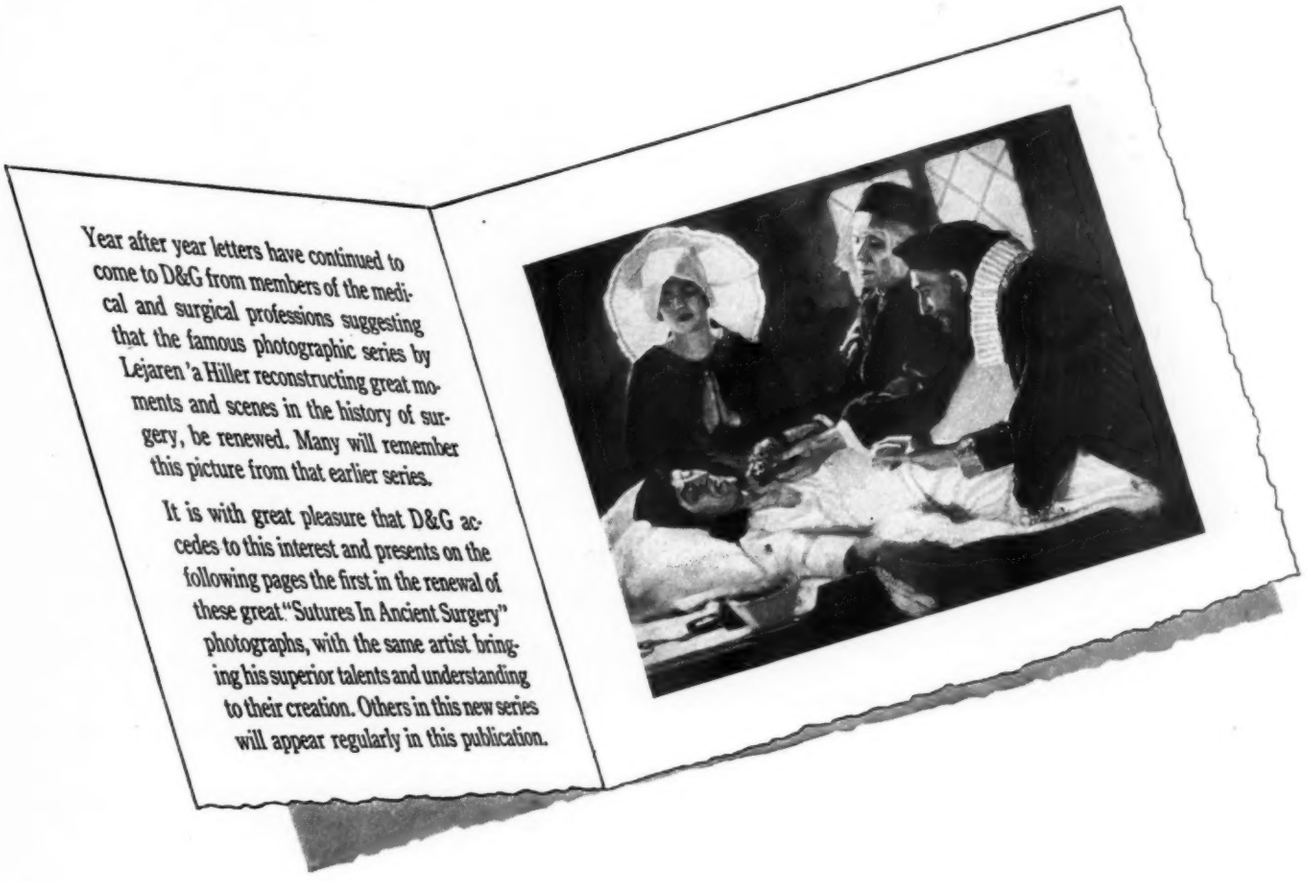

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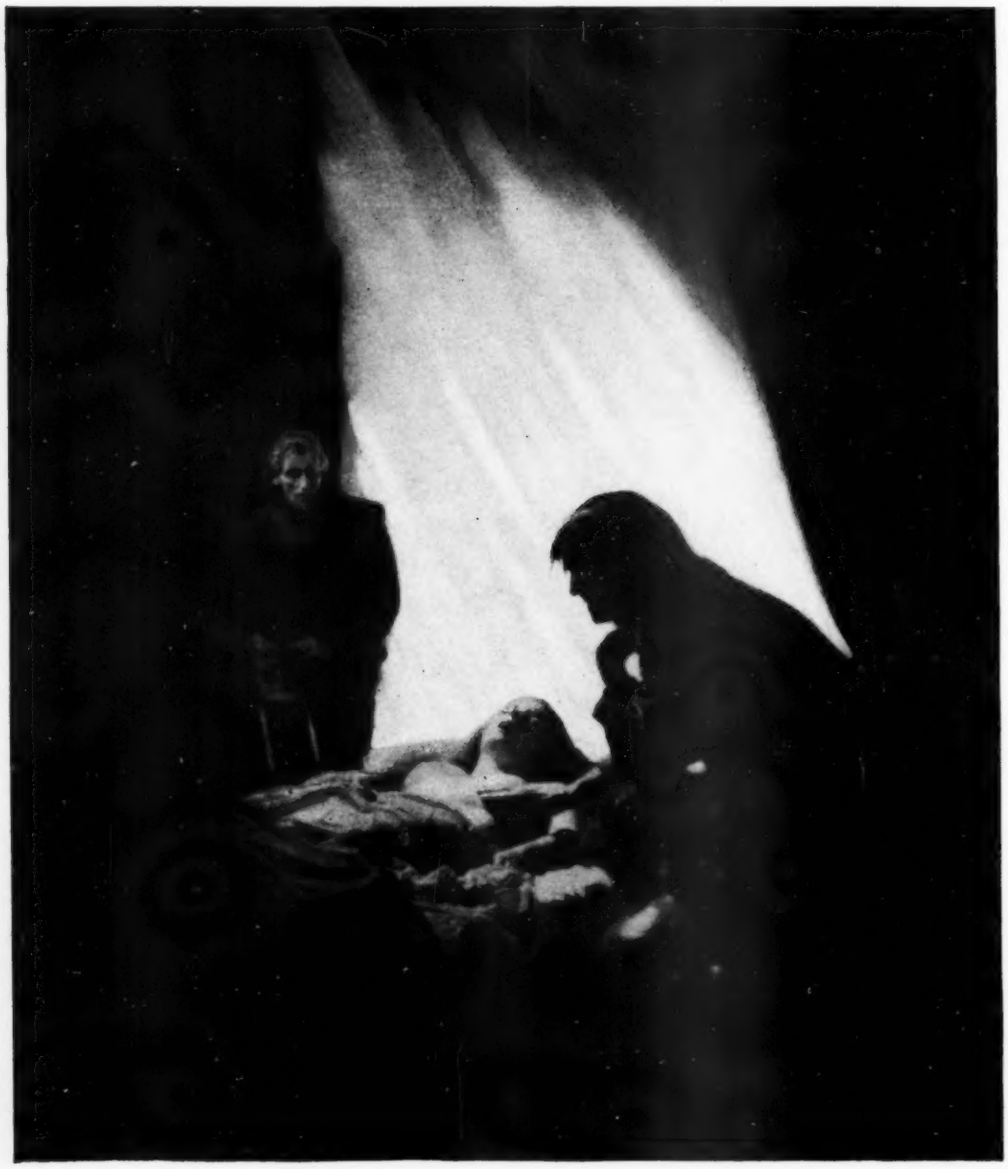

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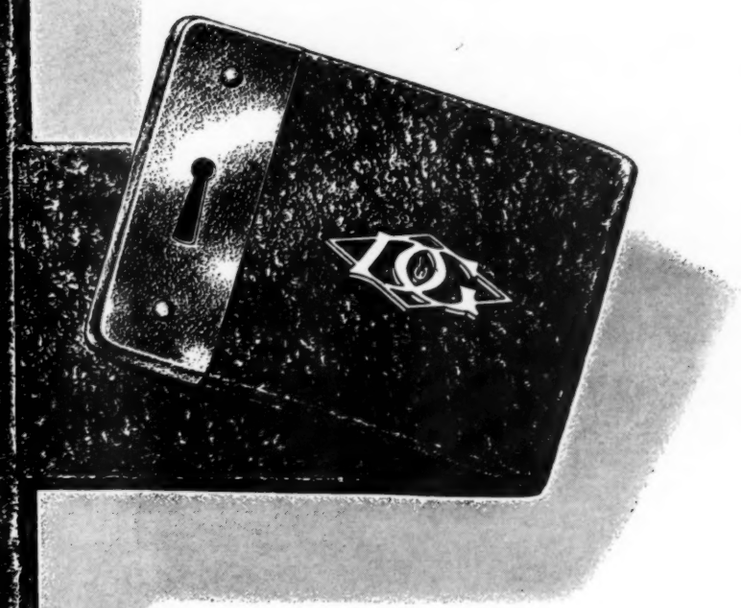

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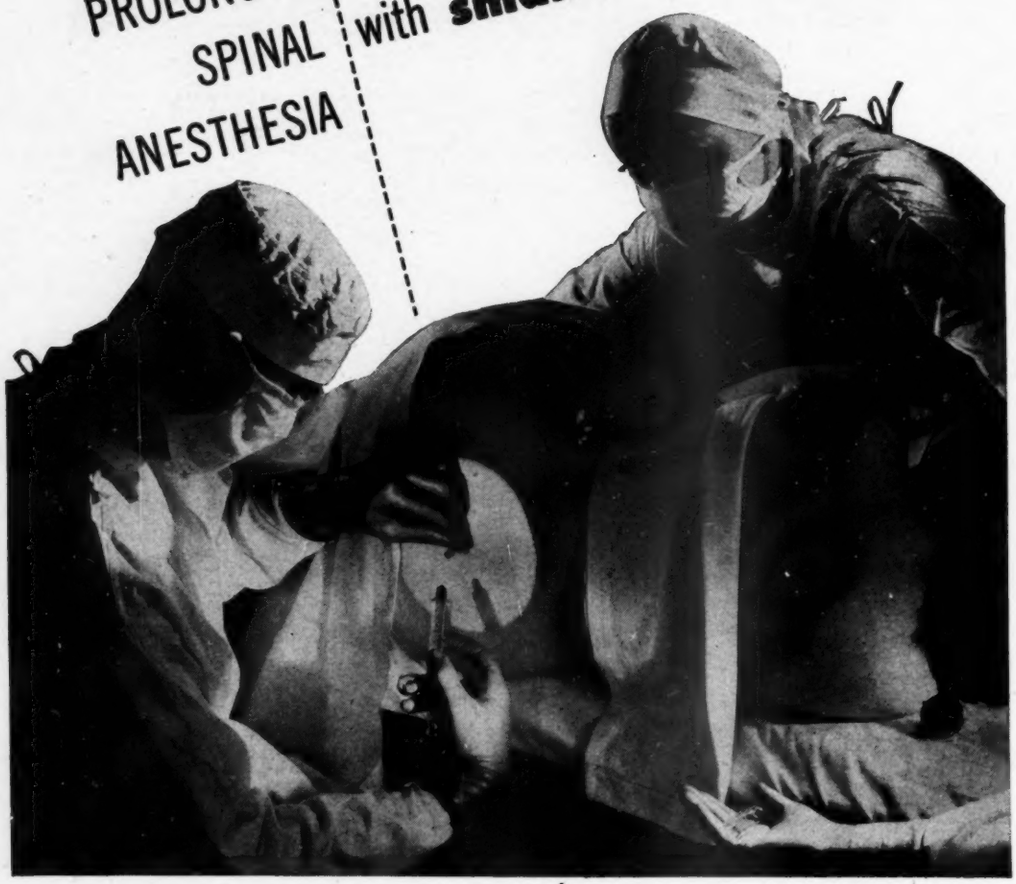

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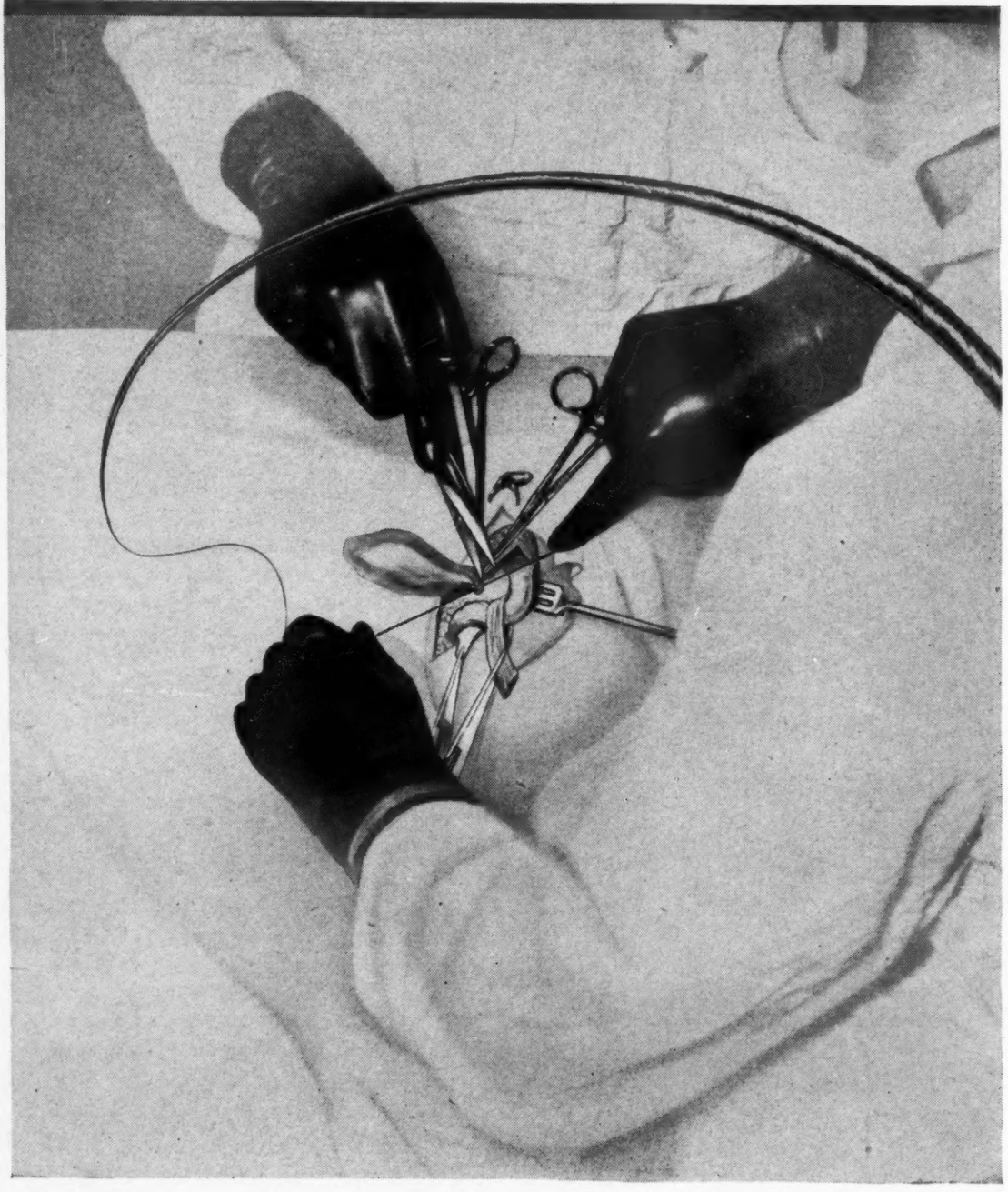

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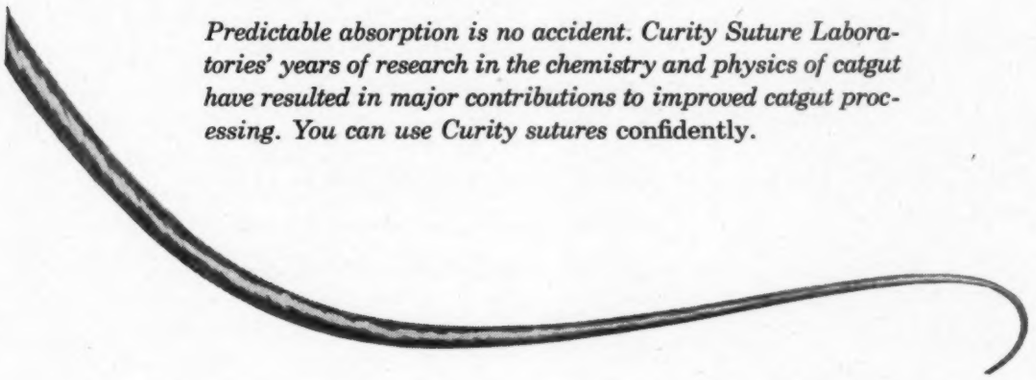

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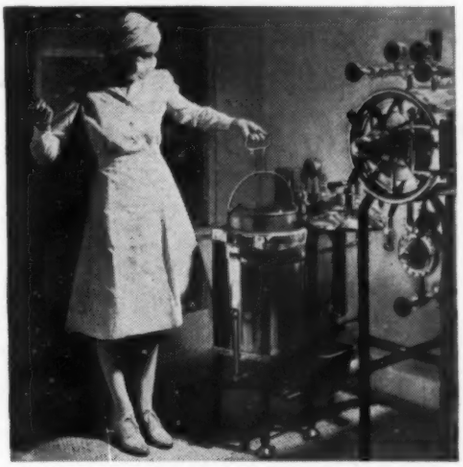

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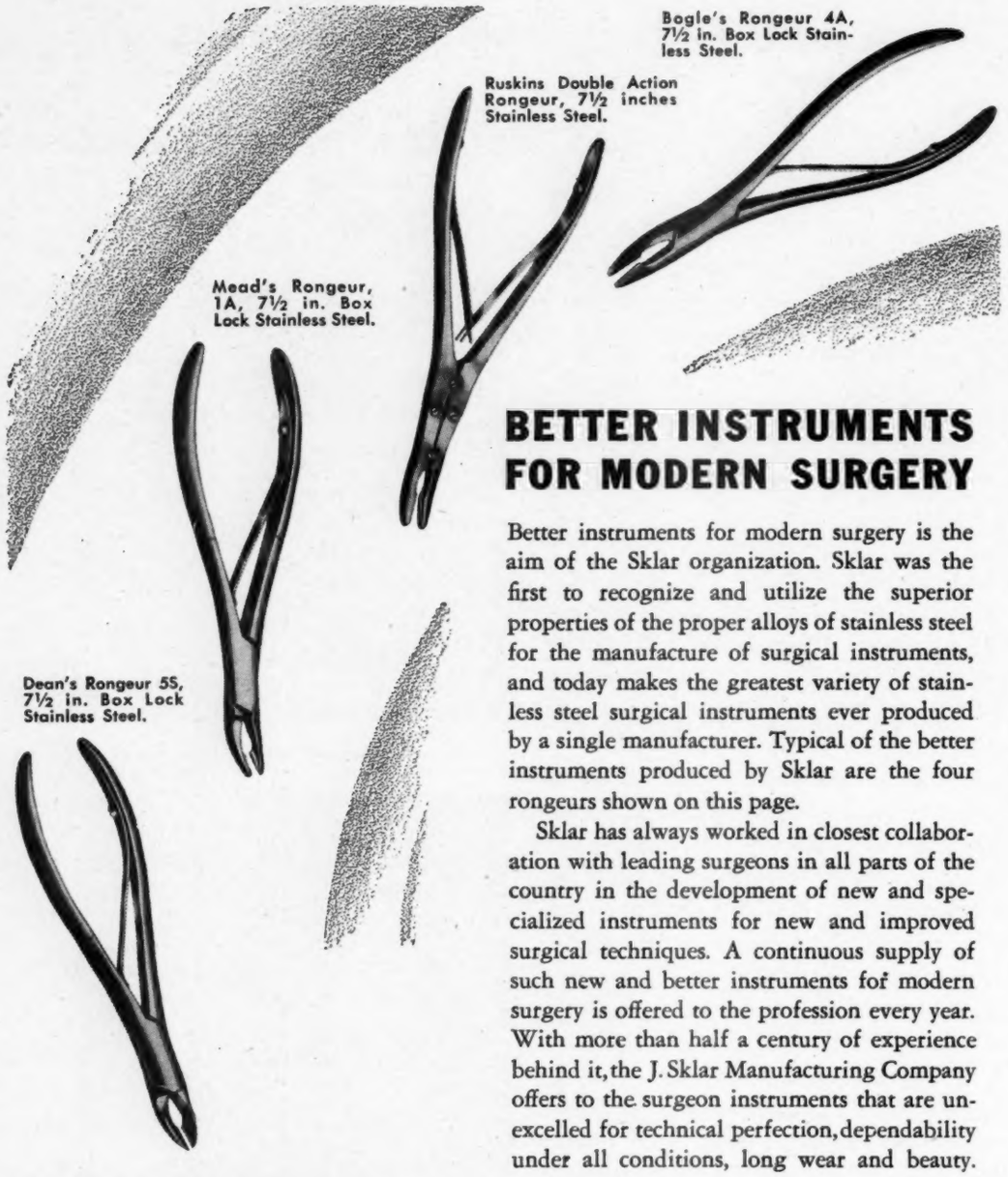
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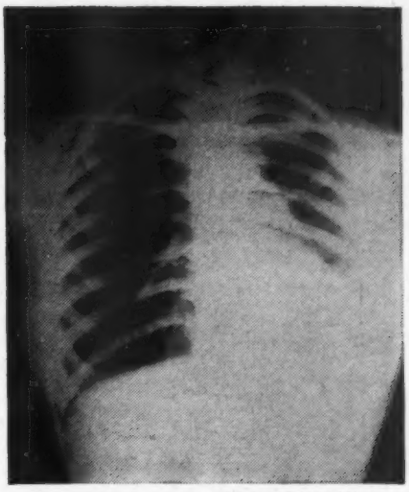

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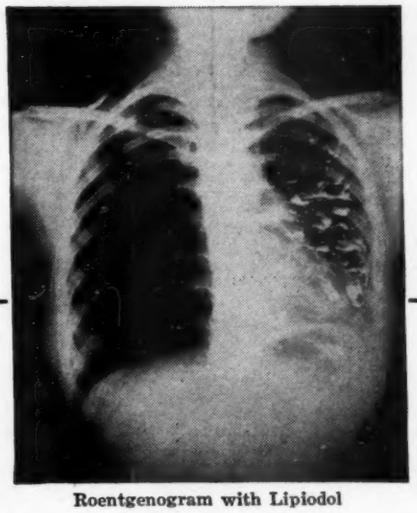

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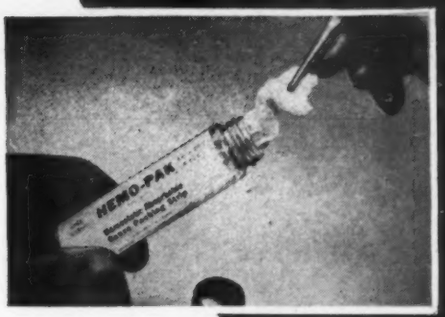

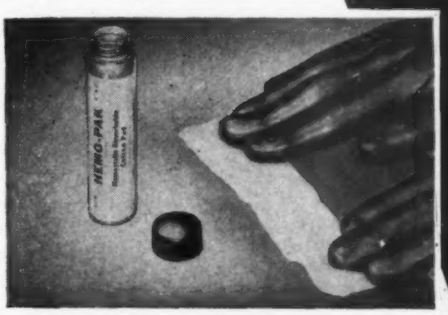

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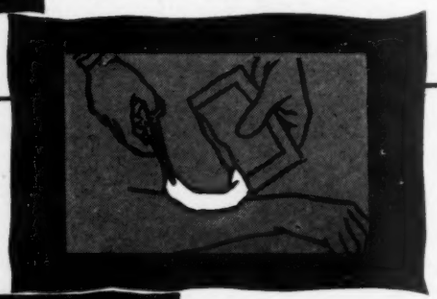

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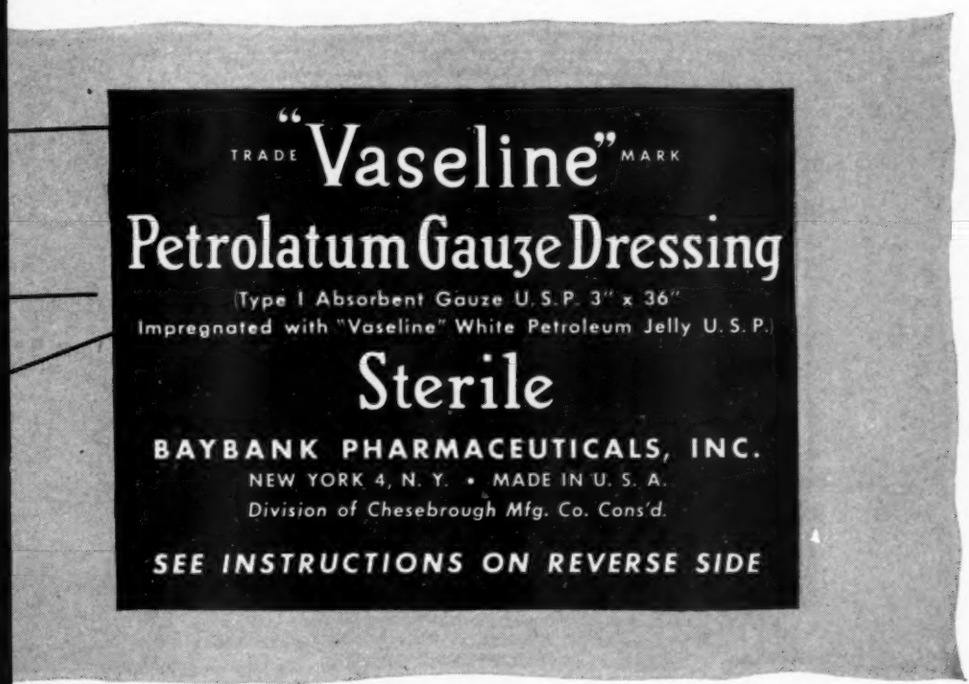
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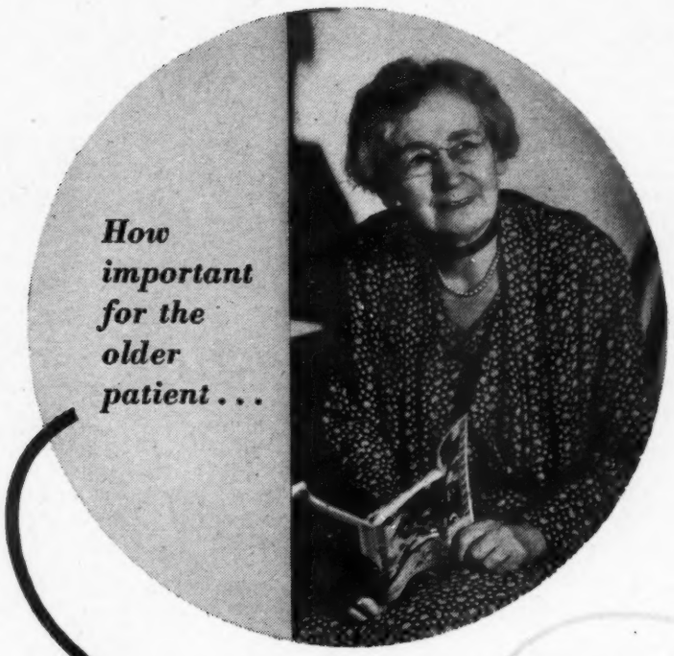

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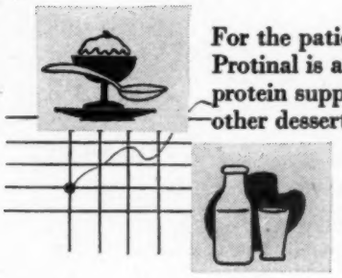

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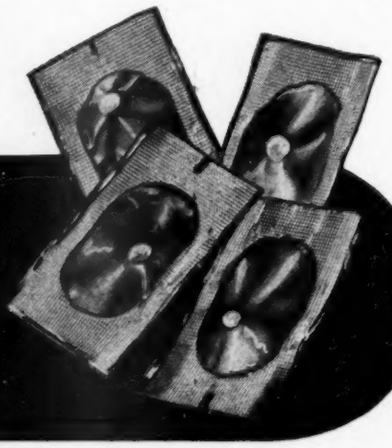

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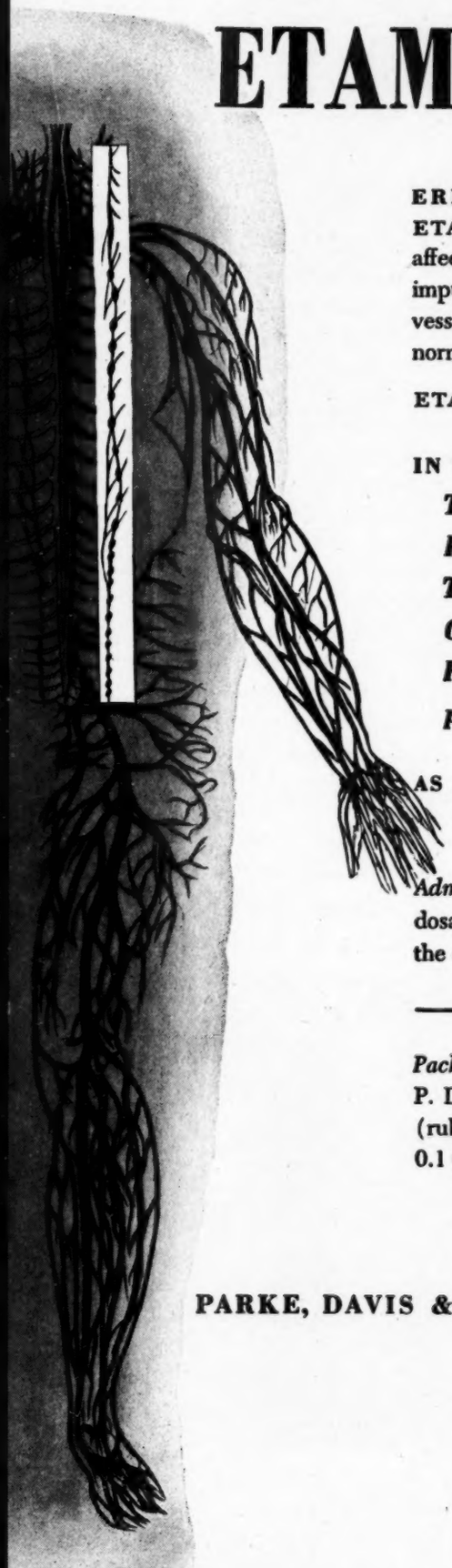

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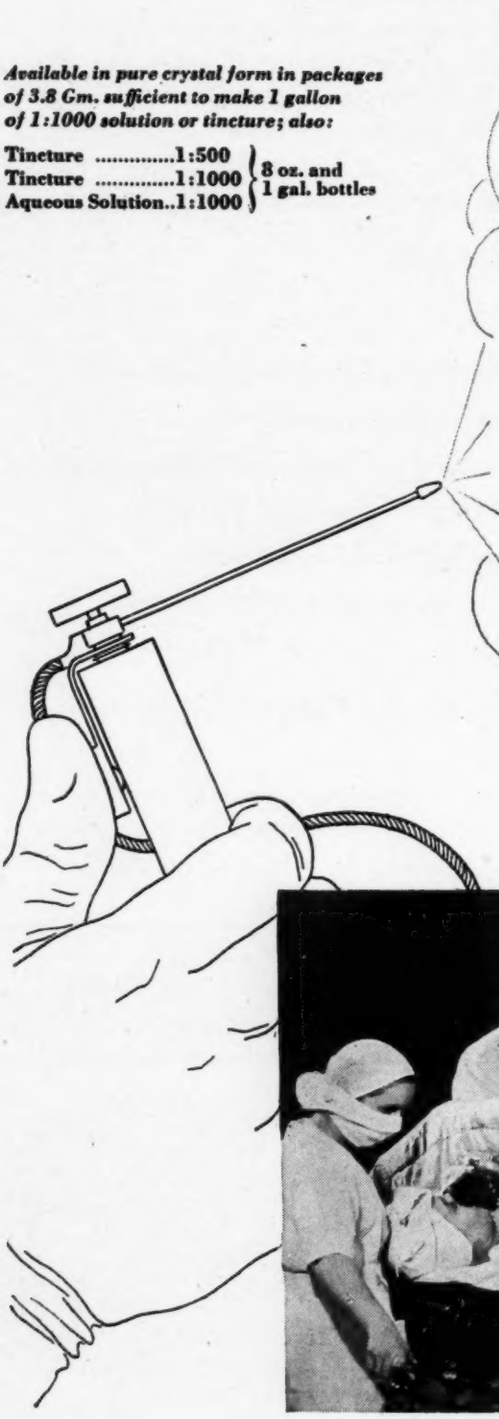

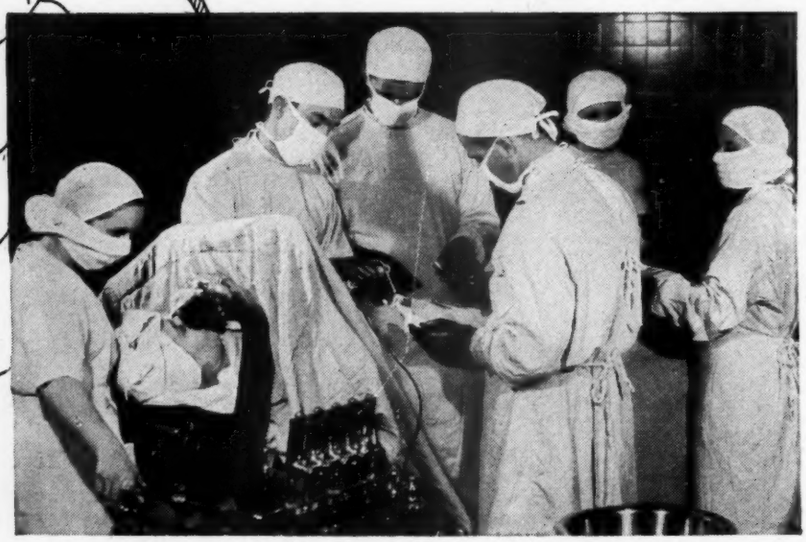

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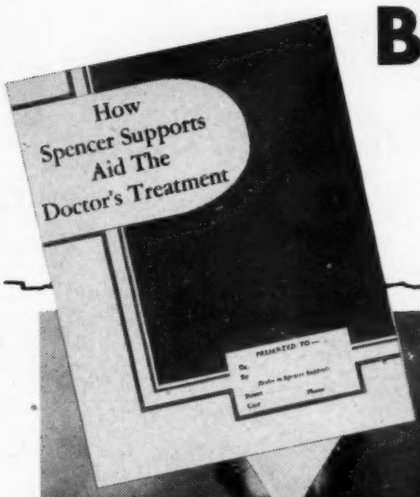

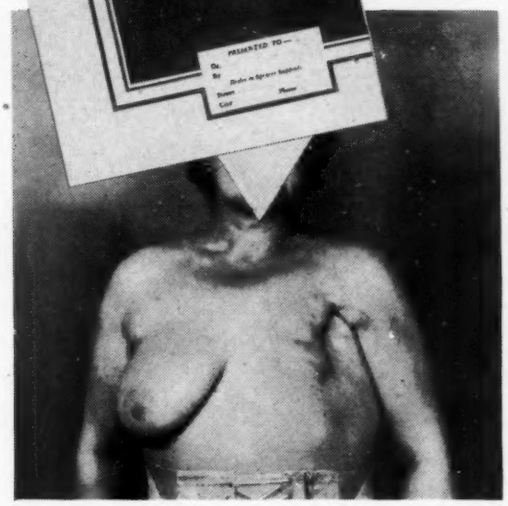

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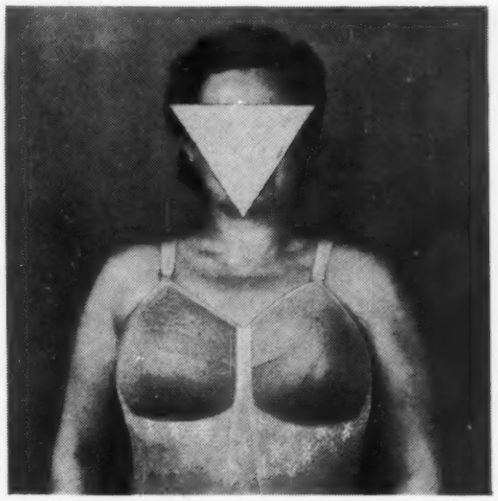

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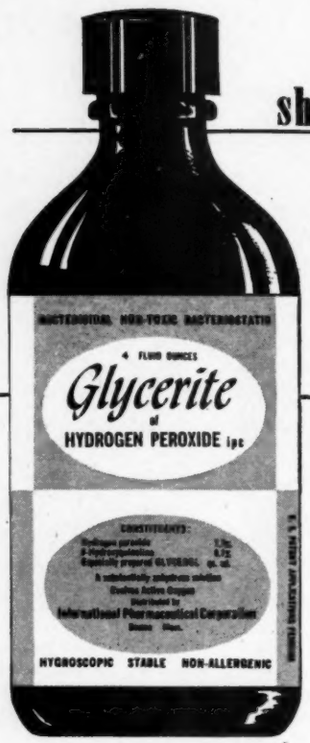

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1. Troup, G. R.: M. J. Australia 34:259 (Aug. 30) 1947.
2. Knight, R. T.: Canadian M. J. 55:356 (Apr.) 1946.
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4. Baird, J. W.: Anesthesiology 8:75 (Jan.) 1947.

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1. Apgar, V. (1946), Experience with Curare in Anesthesia, Ann. Surg., 124:161, Aug. **2.** Fakehany, J. P. (1946), The Use of Curare to Lessen Anesthetic Hazards, Anesth. and Analg., 25:208, September-October. **3.** Griffith, H. R. (1947), The Problem of Muscle Relaxation in Surgery, Canadian Med. Assn. J., 56:281, March. **4.** Brody, J. (1945), The Use of Curare in Sodium Pentothal-Nitrous Oxide-Oxygen Anesthesia, Anesthesiology, 6:381, July. **5.** Knight, R. T. (1947), Curare in Anesthesia, J. Indiana State Med. Assn., 40:125, February. **6.** *Idem* (1946), Anesthesia in General Practice, Journal-Lancet, 66:323, October. **7.** Lundy, J. S. (1947), Annual Report for 1946 of the Section on Anesthesiology (Statistics on the Use of Curare and Pentothal Sodium), Proc. Staff Meet., Mayo Clin., 22:357, Aug. 20, 1947. **8.** Baird, J. W. (1947), Pentothal-Curare Mixture, Anesthesiology, 8:75, January. **9.** Ostlere, G. (1947), Use of Curare in "Poor Risk" Patients, Brit. Med. J., 1:448, April 5. **10.** Harris, H. E., et al. (1947), The Use of Curare and Pentothal Sodium in Endoscopy, Cleveland Clin. Quart., 14:90, April. **11.** Smith, S. M. (1947), The Use of Curare in Infants and Children, Anesthesiology, 8:176, March.

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3. Herbst, W. P. and Weinstein, J. J.: J. Urology, 51: 325, 1944.

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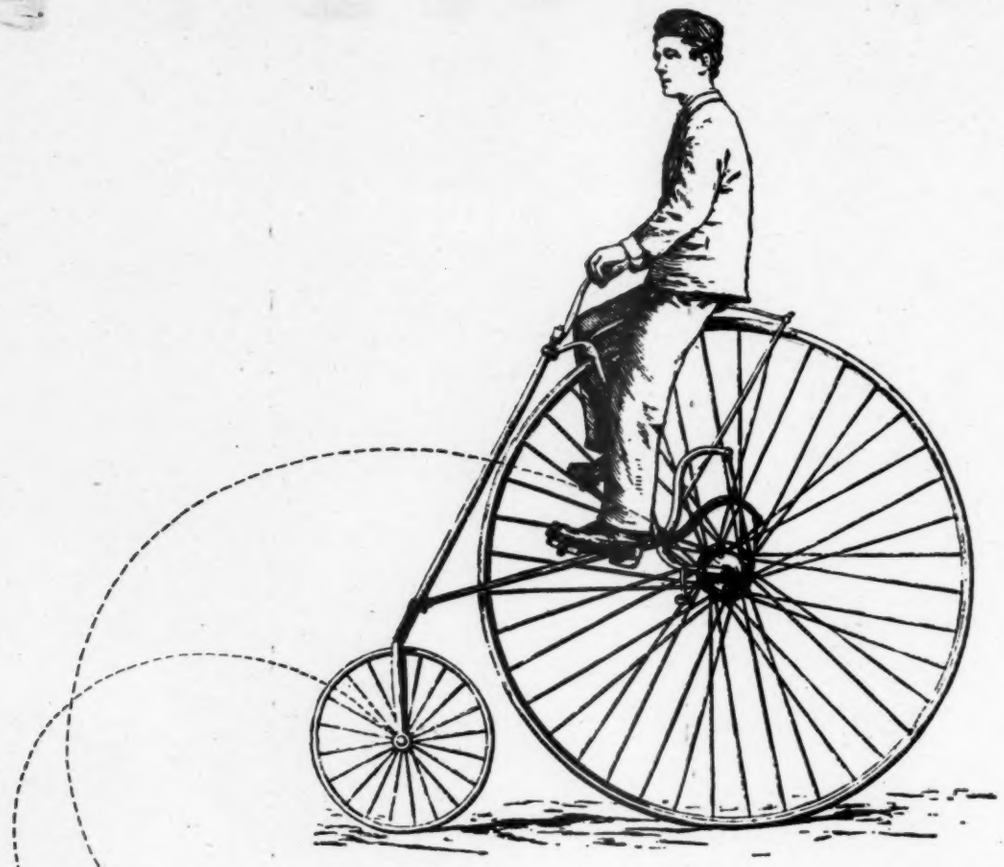

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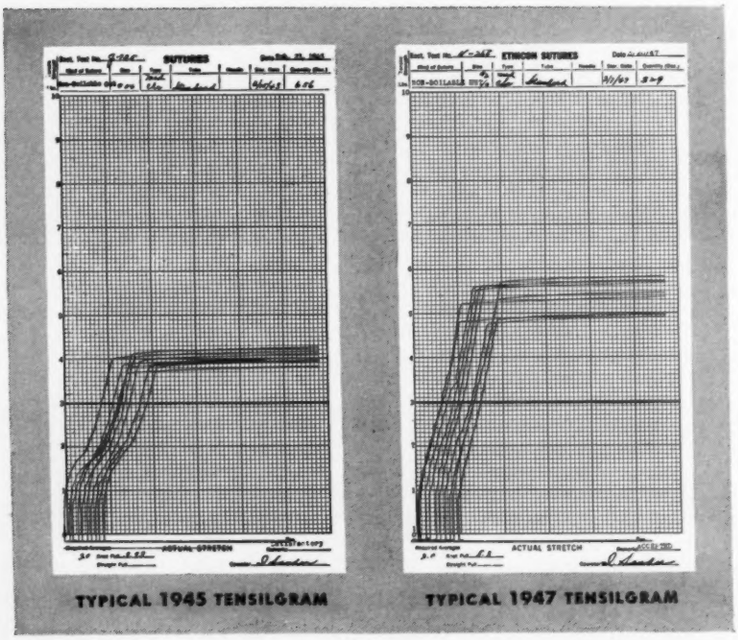

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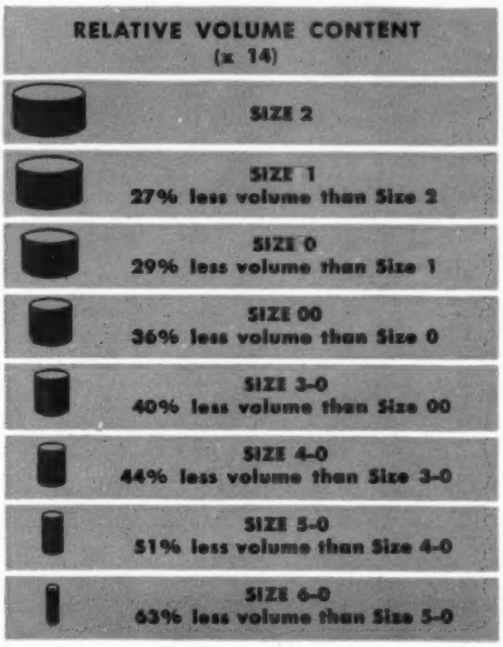

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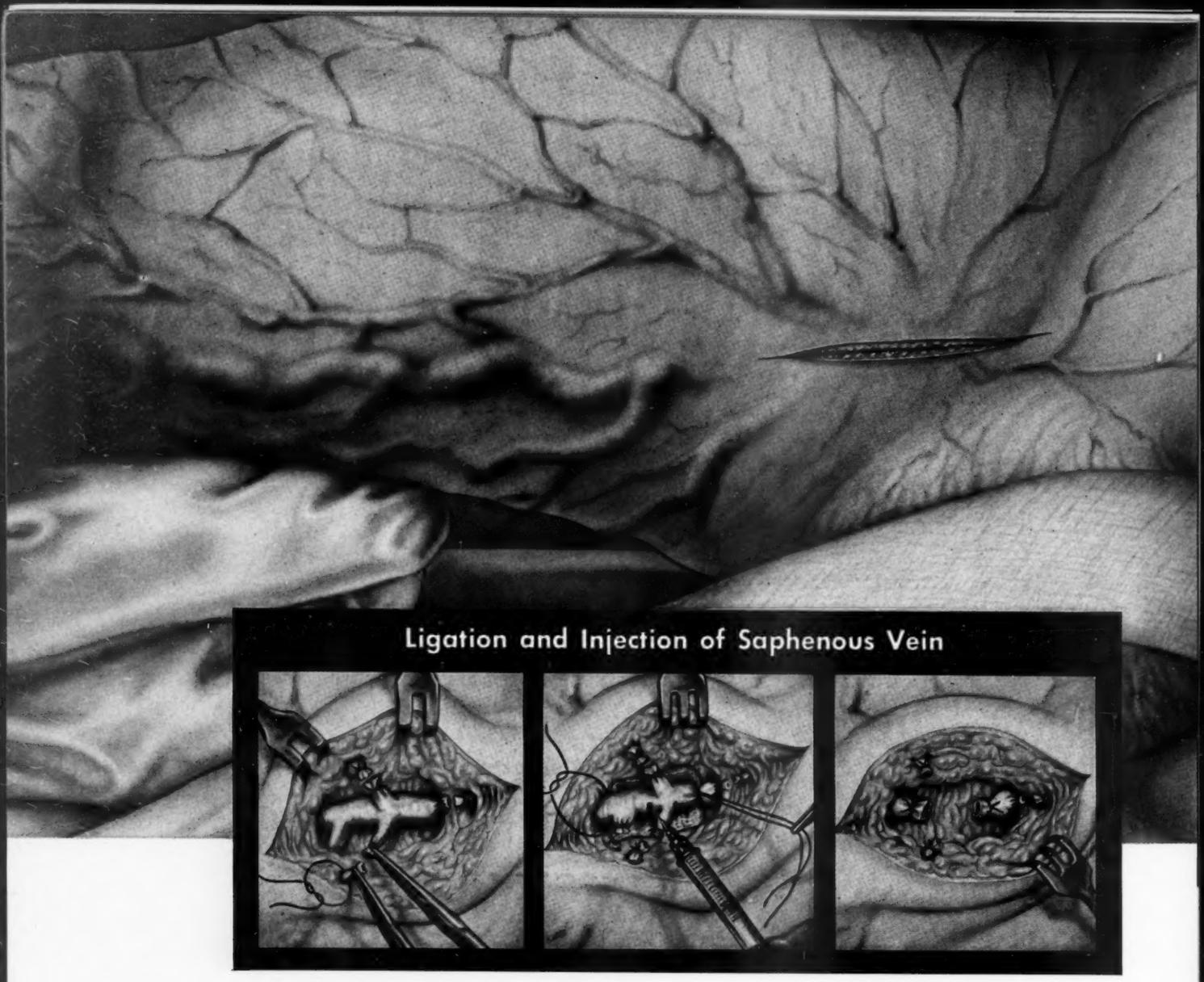

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A PRACTICAL JOURNAL BUILT ON MERIT

Fifty-seventh Year of Publication

VOL. LXXV FEBRUARY, 1948 NUMBER TWO

Presidential Address

THE AMERICAN PROCTOLOGIC SOCIETY

JOSEPH W. RICKETTS, M.D.

Indianapolis, Indiana

AS we come to the close of another year in the history of our society, it is natural and fitting that we should pause to consider how much we have accomplished of what we set out to do and the means to achieve those things we have left undone. In approaching such an appraisal it is tempting, particularly for one of my age, to become reminiscent or to dwell at length on the future. Let me assure you this is not my intention. Many years ago Sir William Osler advised that man's life span should be contained in three lock boxes, one labeled "The Past," the second, "The Present," the third, "The Future," and that only the one labeled "The Present" was ever to be opened, on the theory that if one concerns himself diligently with the present, the past and future will take care of themselves. It is this pattern, the present, the immediate present, which concerns us.

It is apparent that times have changed since the organization of our society in 1899. The placid days when we had time to work out our own salvation are gone. We find ourselves living in an era of racial, religious and political confusion and unrest, with recurring wars and rumors of war that threaten the annihilation of the peoples of the earth. Our own government tended to go leftist, repudiating many of the principles upon which our democracy was founded and instituting bureaucratic regimentation in many phases of public relations, including our own profession. Any conclusions concerning our standing as physicians or special groups of physicians must take into consideration the effect these changes have had and will continue to have on the practice of medicine.

One who has had the honor and distinction of being president of this society for a year must of necessity have been impressed with the problems which confront our organization, a fact no doubt which explains the time-honored custom of the president addressing the society at the opening of each annual meeting. With this thought in mind, I would like to submit some of the important questions that have thus impressed me as being vitally in need of immediate solution.

MEMBERSHIP

The American Proctologic Society was conceived by a group of able, conscientious physicians who set out to establish proctology as an organized specialty of medicine. Through the succeeding years since its founding, the tradition and influence of this group have reached out like the ripples on a pond where a pebble had been tossed, touching the professional life of every member of this society. During the war doctors had an opportunity to see and evaluate the work of trained proctologists. Military patients, too, became aware of the difference between trained and untrained men in this field, all of which helps to explain the present growing interest in our specialty.

The most valuable asset of any organization or society is its membership. The American Proctologic Society has been a conservative organization but to keep abreast of its growing need, we have seen fit from time to time to increase our membership. At present there are over two hundred active members, the largest enrollment in the history of our society. We have reason to be proud and to believe this is the beginning of an expansion which will see our total membership equal that of the other organized specialities. In this connection I would warn you of the danger of carelessness or indifference in the selection of prospective members. So far we have kept the faith of our forebears by zealously adhering to the requirements for membership as set forth in our constitution which provides for membership by application. As long as we continue to adhere to our constitution we may be assured of the future. However, I firmly believe that we have reached a period in our growth when membership by invitation might better serve our society and better maintain our high standard of membership.

RECOGNITION OF PROCTOLOGY

This year marks the attainment of one of the most cherished ambitions of this society—recognition by the Council on Medical Education and Hospitals of the American Medical Association of proctology as an organized specialty of medicine. In a communication received by the chairman of our educational committee. Dr. Louis A. Buie, from Dr. B. R. Kirklin, the secretary-treasurer of the Advisory Board for Medical Specialties, Dr. Kirklin says:

"This is to notify you officially that the Advisory Board of Medical Specialties at its annual meeting held in Chicago on February 9, 1947, recommends to the American Board of Surgery that those desiring certification in Proctology *only* be certified in that field without having to take the entire examination in General Surgery, but, that those who wish certification in Proctology and Colon Surgery should continue to take the examination in Abdominal Surgery."

Dr. Buie also stated, "This is not all that we asked for, but I believe that it indicates that we are making some progress."

In an evolutionary growth such as we are making perhaps it is just as well we do move slowly, with time to adjust ourselves to the drastic changes incident to full recognition and the establishment of our own board, which are our ultimate aims. Thus, it will be seen we have by no means reached the top of the ladder. There are many and difficult problems to be solved and no doubt some tough sledding ahead before we reach this goal.

In the past there has been a fundamental requirement that every Fellow of the American Proctologic Society have good training in general surgery and proctology while no stipulation has been made as to the length of time required for this training or the conditions under which it was obtained. The caliber of the men who have composed our membership, the quality of their work and the exemplary conduct of our society have given our organization a prestige that has enabled us to press for and gain the recognition our specialty deserves. However, in the future, Fellowship in the American Proctologic Society must

be restricted to those men who are qualified to meet the newer requirements this recognition automatically demands.

Associate membership should be limited strictly to ethical practitioners, only those considered who are truly interested in proctology and who have sufficient training to be eligible to Fellowship when the time comes, that is, to men of ambition, with something to give to the society. We should not be content with a large part of our membership consisting of those satisfied to remain as associates or those seeking associate membership only for self-aggrandizement. Our society must continue to be interested in quality, not quantity.

RESIDENCIES

It is our responsibility to provide the facilities for the training required by the American Board of Surgery, namely, approved residencies. At first glance this would seem to be a simple matter. In reality it is one of the most difficult tasks that has ever confronted this society. First, there are not enough hospitals of approved standing to furnish sufficient residencies to meet the demand. What few residencies were available have been taken over by returning servicemen with priority based on previous service in said hospitals. Second, most of our hospitals, in an honest effort to cooperate in this emergency, have taken on more residents than they have work for and this has resulted in complaints from the residents that they are idle and not getting the experience they need. Third, with the exception of a few large institutions, the majority of hospitals state that they have not sufficient proctologic work to justify a resident in proctology. Fourth, another serious obstacle in the way of securing residencies is the attitude of the general surgeon toward proctology. The chief of the surgical staff of the hospitals which we must contact is usually a general surgeon, whose attitude only too frequently is that he is qualified to do all the necessary proctology. Therefore, there is no need for a specialist or a resident in this field in his hospital. However, by proving to the general surgeon that we can do better proctology than he is doing, through better preparation, we have gained some recognition and cooperation from this group, as exemplified in the endorsement of proctology by the American Board of Surgery. This recognition by the surgical chiefs and surgeons who compose the surgery staff is a necessary step in obtaining the training facilities we need and constitutes an added responsibility for this society to continue to make good. Furthermore, many of the hospitals that we are asking to create new residencies in proctology have no well organized proctologic service. This is our opportunity. It is up to us to raise the standards of proctology in these hospitals or whenever the opportunity presents itself and by so doing create a demand for residencies.

A campaign should be launched to create dispensaries and, later, proctologic services in non-recognized hospitals located in cities and towns where Fellows of the American Proctologic Society are available to take over the responsibility of organizing and training interns and residents. I know of three hospitals which previously had no certified residencies where this was done by other specialties. Later, these services were approved and certified by the examining board of the Council on Medical Education and Hospitals of the American Medical Association. If other specialties can succeed in this manner, so can we. This is a gigantic task to be taken seriously by those who are assigned this work. I would urge that the committee fostering these residencies in proctology continue its contact with the Council on Medical Education and Hospitals with greater activity for it is not fair for us to require training with no available facilities to provide this training. I further would urge each member of the American Proctologic Society to dedicate himself to the task of helping in this crucial matter, that he consider himself a committee of one to spread the propaganda of good proctology in his state and local medical societies,

in his hospital staff society and among the interns, residents, nurses and his private patients for there is nothing as effective as united effort.

PROCTOLOGIC JOURNAL

On numerous occasions it has been proposed that this society publish a proctologic journal but nothing has ever been done about it. Formerly, these proposals were made when the need for a journal was not as apparent as it is today, when our society was small and the expense of such an undertaking did not seem in keeping with our financial standing. However, the most likely explanation is that societies, like individuals, hesitate to depart from established custom. If we are to keep in step with the acceleration of our day, it is time we seriously consider the advantages of a monthly, bi-monthly or quarterly proctologic journal instead of being satisfied with the outmoded publication of our yearly transactions.

First, we are a national organization. We encourage the formation of city, group and sectional societies, yet we have no cohesive intercommunication between these groups and the parent organization, the American Proctologic Society. Political, labor, church and other societies have demonstrated the value and need of united effort. A journal would provide this missing link in our national society.

Secondly, our membership meets once a year with no contact between the meetings except occasional terse announcements from our secretary. At each executive meeting vital measures concerning the policies of our society, about which our membership has had little or no previous knowledge, are presented and voted on. If we had a journal, these questions could be publicized, giving each member time to formulate a just opinion. This would minimize confusion, lessen the chance of mistakes and promote better understanding which makes for a better society.

Third, for those who attend our yearly meetings the material in our transactions is dead copy—a reference book only. Papers of value are being read before groups and sectional societies through the year that should be available to our membership at large. For example, I had the pleasure of attending the March meeting of the Philadelphia Proctologic Society. Three excellent papers and a review of the literature for the previous month were presented. Each of the papers read were of national society caliber and could have been accessible to all of our members if we had a journal. Yet I dare say only those present will profit by what was presented.

Fourth, our annual program of necessity is limited in the number of papers to be presented. This excludes many who would like to appear on the program and many who should appear. A journal would remedy this difficulty by providing a means for surplus new papers to reach the membership and it would encourage the new members, with little experience in writing papers, to develop this art.

Fifth, our honorary members, composed of men of outstanding proctologic knowledge, could be invited to contribute papers to appear in our journal. This would not only be another source of valuable material but would provide contact with a group of our society which has been neglected in the past.

Sixth, publication of announcements, obituary notices and open letters addressed to officers of the society, chairmen of committees or individual members on professional, political, legislative or personal problems, would make for a better informed membership.

Seventh and finally, the establishment of a Journal of Proctology is imperative in order that the aims and importance of the American Proctologic Society may receive full recognition. Had we possessed a journal such as is proposed during the past ten years, our struggle to place proctology in its rightful place with the other recognized specialties of medicine would have been accomplished in less time and with more decision than has been the case.

Having stated but a few of the arguments for a journal, and I dare say each member present could add to this list, the next consideration is the expense involved in such an undertaking. Last year our transactions cost this society $4,800.00.

An estimate from two reputable publishing houses on 300 monthly copies of a journal of one-hundred pages, paper and type to be the same as used in the average state medical journal, was $4.00 per page, or $400.00 a month, or $4,800.00 a year. Some of this cost would be absorbed by revenue from advertising. The printing bill, labor and postage incident to the reports and announcements sent out by our secretary is an item that could be deducted from this cost as such material would appear in the journal. Thus, it will be seen that the actual cost of publishing a journal would not materially exceed what we are expending for our transactions. There will be additional costs, of course, but the advantages to be had will be well worth the added investment.

The far-reaching influence of a Journal of Proctology upon the future development and growth of the American Proctologic Society, both within its own organization and in its contacts with other branches of surgery, must be evident to all of you.

FINANCIAL STATUS

It is an established economic fact that the cost of operation increases inversely with the expansion of a business or organization. The truth of this maxim was brought home to the Council of the American Proctologic Society at its Miami meeting. Here our treasurer announced we could no longer operate our growing society on the present hand to mouth financial basis; some new source of revenue would have to be found to carry on. The Council's reaction to this statement explains the unpleasant news you will receive in the form of an increase in dues and initiation fees, but this in itself is not sufficient. A growing society needs reserve capital for new projects which are a part of any expanding program.

In discussing with our secretary ways and means to build up such a reserve fund he offered this pertinent suggestion—that we interest our members in providing for the American Proctologic Society in their wills. It occurred to us that since gifts are deductible from income tax, our past presidents might be interested in making a direct gift which would be available to the society for immediate use. Also the membership in general might be interested in some form of life insurance with the American Proctologic Society as the beneficiary. This is done frequently by individuals who desire to leave something to a church, lodge, fraternity or some other organization. For those of our members who might be interested in this plan ordinary life insurance, with the society as beneficiary, could be purchased on their lives to cover any amount they wished to designate for a nominal annual premium. Such a plan would give those members the assurance and satisfaction that at their death the society would receive a legacy from them that would help build the future financial stability of the American Proctologic Society.

The foregoing suggestions are made merely to show the way for a new, sound financial policy with a definite budget system, which I maintain is imperative if we are to go forward.

SUMMARY

In closing, I wish to pay tribute to the sincere, active Fellows of the American Proctologic Society whose prolonged pioneering efforts in the years past, since the inception of our society, have been responsible for the remarkable achievement in the proctologic progress that has been attained. Time does not permit giving mention of those Fellows. Suffice it to say their names will always be a source of inspiration to those who will carry on. Whether or not this progress shall be sus-

tained is solely dependent upon the future activity of our younger Fellows.

As president of the American Proctologic Society I want to thank the officers, members of the Council, those who have served on committees and our genial and efficient secretary and his secretary for their earnest consideration and cooperation during the past year in making, what at first seemed to me an insurmountable task, one of the most pleasant experiences of my professional career. I wish to express my appreciation to those of our members and guests who are contributing of their time and energy by appearing on our program. Also I want to thank the wives and sweethearts of our members for their gracious presence which always has added so much to the success of our meeting.

Last but not least, I want to express my sincere thanks to the members of this society for the privilege and honor of serving as your president during this past year. As retiring president, I pledge my best effort to the administration of our president-elect, with a prayer that 1948 will be one of the most successful years in the history of the American Proctologic Society.

Prolapse (rectal) is a condition of abnormal downward mobility or laxity of mucosa membrane. Actual protrusion through the anal orifice is not essential, for the prolapse may involve the mucous membrane lining of the sigmoid of the rectum and be obvious only upon digital or endoscopic examination.

Case Reports

MULTIPLE MALIGNANT LESIONS OF THE COLON

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NUMEROUS cases of multiple primary, malignant lesions, have been reported in the literature. In an article published in 1945, Bacon[1] gave a complete summary of cases reported to that date. The case herein reported is considered of interest as this patient had a right colectomy for carcinoma ten years before presenting herself with a far advanced lesion of the sigmoid which required a segmental resection and hysterectomy. An end-to-end anastamosis was subsequently done with restoration of normal function.

CASE REPORT

This patient, a sixty-nine year old white female, entered the hospital October 28, 1945, with a primary complaint of progressive weakness, the onset of which was two or even three years prior. This complaint had become markedly worse the last six to eight months before admission. Three days before admission she noticed blood in her stools for the first time since her former admission ten years ago. She had noticed dark but not tarry stools. She gave a history of alternating diarrhea and constipation for several months, but there had been no nausea, vomiting or any symptoms of obstruction and she had been in no pain since her former operation.

She had had rheumatic fever and the usual childhood diseases. In November, 1935, the terminal ileum, cecum, ascending and proximal one-third of transverse colon were resected and an ileocolostomy made, using the Murphy button, for a fungating annular adenocarcinoma of the cecum, followed by an uneventful recovery.

Physical examination revealed a thin, pale, emaciated white female of sixty-nine. Her head, neck, lungs and thoracic cage were essentially normal. The heart beat was rapid (120) regular; no abnormal sounds were heard. The abdomen was protuberant with a midline scar; there was no rigidity or tenderness. There was a suggestion of a mass in the right lower quadrant with a definite mass in the left abdomen, giving the impression of a colon filled with feces. The extremities and neuromuscular system were essentially normal.

Laboratory findings were as follows: Numerous specimens of urine showed occasional pus cells; red blood cells, 3,590,000; white blood cells, 11,800; hemoglobin, 11 Gm. 65 per cent; differential and Schilling counts normal; clotting time three and one-half minutes; urea nitrogen 14 mg., occult blood in feces.

At the request of the medical section, the patient was seen by the author on October 30, 1945.

Proctoscopic examination revealed combined hemorrhoids grade 1 and no evidence of bleeding from any lesion at the ano rectum. The rectal mucosa and valves appeared normal. At 20 cm. two discreet adenomas were noted about 2 mm. in diameter which were removed for biopsy. The bowel was fixed and angulated at 24 cm., a large amount of blood-tinged mucus was aspirated, but the lumen could not be scoped with a small diameter scope because of its fixation.

X-ray examination on October 31st was reported as follows: "Injection of barium causes considerable distention of rectal ampulla and distal sigmoid. Complete obstruction is encountered in the region of the right lower quadrant and there is evidence of colonic delay above this point. *Conclusion:* Obstructive lesion of distal sigmoid probably malignant."

The patient was prepared for operation but declined, believing she was unable to stand a

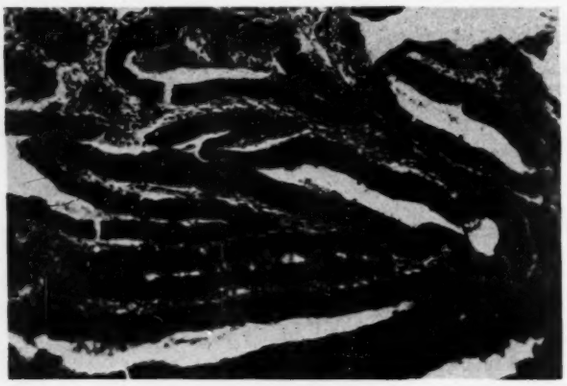

Fig. 1. Photomicrograph of lesion showing microscopic appearance of lesion.

second operation. However, she then consented and was operated November 8, 1945, under spinal and pentothal sodium anesthesia. The abdomen was opened through a midline incision. Upon opening the abdomen, exploration revealed a large mass in the pelvis. The liver was palpated and found to be free of any evidence of metastasis, and no nodes along the course of the great vessels could be palpated. It was found on further exploration of the mass that the uterus and the sigmoid were adherent with an extension of the malignant lesion into the uterus. It could not be separated without breaking into the septic mass, and for this reason it was decided to do a hysterectomy and resection of the sigmoid as one unit. This was done by first dividing the round ligaments and Fallopian tubes bilaterally, which were in turn individually ligated with No. 1 chromic catgut suture. The dissection was carried around posteriorly between the peritoneal folds. The uterine arteries were identified and tied with suture ligatures of No. 1 chromic catgut. The uterus was then amputated, the cervical os was cauterized with tincture of iodine, and the cervical stump closed with interrupted chromic catgut sutures. The uterus was thus freed from its cervical and ligamentous attachments, but adherent to the colon. The bowel was then mobilized by freeing the peritoneal fold laterally, then medially and ligating the sigmoidal arteries and veins with No. 1 chromic catgut. The bowel was divided proximally above the mass and divided distally below the mass by use of the DeMartell clamp. A colostomy was contemplated with the proximal portion of the descending colon, but it was decided that the rectum could be mobilized. This was done by freeing attachments laterally and its loose fascial attachments posteriorly, and bringing up the rectal stump. The two spurs of the colon were then approximated with a Rankin clamp. Sulfanilamide powder was loosly sprinkled retroperitoneally and intraperitoneally. The pelvic floor was peritonealized and the abdomen was closed in layers, using plain catgut to peritoneum, interrupted chromic to fascia and dermal to skin. Steel wire stay sutures were used. The patient was then turned on her right side and a drain placed into the retrorectal space, up beneath the posterior peritoneum of the abdomen to drain the area of oozing blood due to the freeing of the rectum from the intraperitoneal route.

A report of the histologic examination of tissue removed is as follows: "One specimen consists of several lymph nodes and a mass of hemorrhagic tissue, which grossly does not appear remarkable. Also present is a loop of large bowel which measures 37 cm. in length. The bowel is greatly distended. On section, the distention is found to be due to a tremendous tumor mass, which measures 8 by 9 cm. It protrudes 3.5 cm. from the wall of the bowel. Also present is a corpus uteri, which measures 6.5 by 5.5 by 3.5 cm. It contains a subserosal leiomyoma. The uterine body itself is not too remarkable. Microscopic sections present a papillary adenocarcinoma of the large bowel. Sections through the uterine wall present evidence of infiltration and there is also marked macrophage activity. The macrophages are unusually prominent. Sections of one adjacent lymph node present early metastatic involvement."

Pathological diagnosis: Papillary adenocarcinoma of the large bowel with obstruction and extention to the uterus and adjacent lymph node.

The postoperative course not remarkable. A Miller-Abbott tube was passed into the small bowel. The clamp to the proximal stoma was opened on the third day. An indwelling catheter was removed on the fourth day. A normal stool was passed from colostomy on the fifth day following a small oil retention enema. On the sixth day the tube was removed and fluids were given by mouth. The posterior drain was completely removed on the eighth day. The patient developed a sudden severe upper abdominal pain on the eighteenth day which subsided after seventy-two hours, the cause of

which could not be determined. She was discharged on the thirty-second day, having remained in the hospital longer than necessary because no adequate nursing home could be found at this time. Her general condition was good and the colostomy was functioning well.

The patient was readmitted to the hospital from the clinic on March 30, 1946. Her colostomy was functioning well, she had gained weight and strength and appeared well nourished. Her physical findings were essentially negative. She was urged to continue with her colostomy, but she insisted upon a closure as she desired to return to her native Sweden. She was prepared for surgery in the usual manner.

She was operated upon April 17, 1946. The colostomy was closed with a black silk purse-string suture after the usual abdominal preparation. The abdomen was entered by excision of the old scar. The abdomen was remarkably free of adhesions. The blind rectum was freed from adhesions and mobilized. The two adjacent ends of the bowel were then caught up in a Rankin clamp and the ends of each cut off with a cautery. A closed anastomosis was then performed, using a posterior and an anterior No. 00 chromic catgut suture in the procedure as described by Rankin. The anastomosis was reinforced posteriorly and anteriorly with interrupted black silk sutures. A long colon tube was passed up through the anastomosis. Sulfanilamide powder was sprinkled lightly in the abdominal cavity, raw surfaces were peritonialized and the abdomen closed. A small rubber drain was left in the abdomen at the operative site, plain catgut was used for the peritoneum, No. 1 interrupted chromic for the fascia, interrupted black silk for the skin, with a small rubber tube drain being placed to the fascia.

Postoperative convalescence was wholly uneventful. The patient passed gas through the rectal tube; no decompression was necessary. The tube was shortened every forty-eight hours and removed on the sixth postoperative day. The patient had two bowel movements the following day. All skin sutures removed by the tenth day. The wound healed without drainage; normal bowel movements occurred and the diet was begun on the sixteenth day. After leaving the hospital, the patient presumably went to Sweden and there has been no follow-up note since.

CONCLUSION

A case of a seventy year old white female, who ten years previously had a right colectomy for carcinoma of the cecum, She returned with a far advanced carcinoma of the sigmoid, involving the uterus. Hysterectomy and segmental resection of sigmoid *en masse* was performed. A delayed end-to-end anastomosis of the colon was followed with complete healing and normal function.

The pathologic picture of the tumor is that of a papillary adenocarcinoma of the colon, most generally encountered in the right colon rather than the scirrhous type usually seen in the left.

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ISCHIO-ANAL DERMOID

JAMES D. SCHOFIELD, M.D.
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THIS case is of interest chiefly for two reasons: dermoids in an infralevater situation are, so far as I could determine, unreported, and although dermoids of the rectum are sparsely reported in the literature, they are almost exclusively found in the female. Dr. Arthur A. Landsman, in 1929, reported the only other case of such a lesion in the male; it was an intrapelvic dermoid pointing into the rectum.

CASE REPORT

Mr. J. A. McG., white, age forty-one, was admitted as an emergency patient to Hahnemann Hospital on November 7, 1946 with the diagnosis of ischio-anal abscess. A painful swelling in the left anterior perianal quadrant had developed approximately seven days before and the size and discomfort of the lesion had been increasing ever since. He gave a history of a similar abscess at the same site in 1939 which had been incised and unroofed. The surgeon had informed him then that the abscess was of an unusual type, being very deeply seated, and that further difficulties might be anticipated. His proctologic history was in all other respects unremarkable. Examination revealed a large, angrily inflamed, fluctuant abscess in the left perianal quadrant.

On the day after admission the patient was operated on under sacrocaudal intracaine anesthesia. The abscess was incised widely with the escape of a copious purulent discharge. After bevelling the margins of the incised abscess, further exploration of the tract in the portion proximal to the anus led to a deepseated pear shaped sac. This sac was dissected and removed in toto. It measured approximately 4 by 2 cm. When opened it was found to be filled with a large nest of hair, necrotic tissue mixed with feces and a fragment of a tooth measuring 0.6 cm. in its greatest diameter. Microscopic examination revealed the composition of the sac to be mostly connective tissue, with clefts lined by stratified squamous epithelium. Because the dissection had required a wound of excessive breadth, incision of the sphincter was deferred, a seton being placed through the internal sinus and the unopened portion of the tract.

The patient's postoperative course was highly satisfactory. By November 21st, thirteen days after operation, the wound had healed sufficiently so that the remainder of the tract could be incised and the seton removed. Two days later the patient was discharged. Subsequent healing was kindly and by November 30th was complete.

In this case the dermoid lay sufficiently close to the midline to lead the author to surmise that its origin was an invagination of skin during coalescence in the perineum. The mode of development of the communication with the anus is a matter of speculation.

In general dermoid cysts are benign. Malignant degeneration, although rare, does occur and may be carcinomatous or sarcomatous, with or without metastasis. If at all feasible, they should be excised in their entirety, not only as a safeguard against infrequent cancerous change, but also to put their harm to an end. In 1938 Dr. J. F. Saphir reported a case of true dermoid cyst of the anterior wall of the rectum which, of necessity, had to be treated conservatively although it occasioned much inconvenience to the patient. In his paper Dr. Saphir included an excellent review of the literature concerned with rectal dermoids, to which little has been added since.

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MESENTERIC THROMBOSIS FOLLOWING THE INJECTION TREATMENT OF HEMORRHOIDS

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In a limited review of the literature I have found no mention of mesenteric thrombosis as a complication of injection therapy. Bacon[1] mentions hemorrhage and slough as the most common complications and ulceration, proctitis, stricture, abscess, fistula, liver abscess and pulmonary infarction as more remote complications.

Rankin, Bargin and Buie[2] mention four patients in whom disastrous consequences resulted from injection therapy; two of their own and two who were injected by others. These cases were all due to massive slough. Yaker[3] lists complications reported in the literature but makes no mention of mesenteric thrombosis. Morgan[4] in his article, "Castastrophies Following Hemorrhoid Injection," reports a case of cellutitis and slough of the entire anus and rectum.

Mabrey and Speare[5] add pelvic phlebitis and pyemia to the list. Rosser[6] adds formation of oil tumors or eleomas. None of the writers mention having experienced, observed or read of mesenteric thrombosis as a complication of this form of therapy.

The purpose of this case report is neither to discredit nor approve injection treatment of hemorrhoids. My policy has always been to limit injection treatment to the uncomplicated internal variety of medium size. If the patient states that his hemorrhoids protrude upon defecation and especially if they require manual reduction, I immediately suspect that they are not suitable for injection therapy.

CASE REPORT

The patient, Mrs. J. P. H., white female, age fifty-eight, was referred by her family physician on September 10, 1946. Her complaint at this time was swelling at the anus which was slightly painful, constant, not reducible and had been present for three days. The pain and swelling was not aggravated by evacuation. No bleeding had occurred nor had there been any protrusion on evacuation.

The past history revealed that she had experienced no severe illnesses, accidents or operations except a minor anorectal procedure performed in the office of a local proctologist four years previously. Her family history was irrelevant.

On examination the patient was well developed, well nourished, quite co-operative and apparently in no acute pain or distress. The abdomen was soft and not distended or tender. The liver and spleen were not enlarged and no glandular adenopathy was palpable. No masses were found. The perianal aspects presented a single external thrombotic hemorrhoid in the left lateral phase less than 2 cm. in diameter. On digital examination the external sphincter was slightly spastic.

Anoscopy revealed internal hemorrhoids of medium size in the left lateral, right anterior and right posterior phases. There was no ulceration.

Under proctosigmoidoscopy the bowel was visible for a distance of 15 to 20 cm. The mucosa was normal in color and no benign, malignant or ulcerative process was present.

Diagnosis: (1) External thrombotic hemorrhoid; (2) internal hemorrhoids, uncomplicated (medium size).

Ambulatory treatment was decided upon because it was thought that the patient's condition was suitable and also because she had an invalid husband from whom she was reluctant to be separated. Accordingly, the external thrombotic hemorrhoid was resected under local anesthesia on the initial visit. Two weeks later, after the external wound had completely healed, she was given her first injection. Five per cent phenol in Wesson oil was used and approximately 1 cc. was injected into the process in the right anterior phase. The usual care and technic advocated for injection

therapy was employed. One week later she reported for her second injection and a similar treatment was given in the left lateral phase.

The patient felt quite well until the evening of October 6, 1946, five days after the second injection when she began to have generalized abdominal cramping. Shortly thereafter she developed nausea and vomiting and her family physician was called. He did not consider her condition alarming until twenty-four hours later when she developed abdominal distention and tenderness in the left lower quadrant. At this time she was admitted to the hospital. There was no fever, the white blood count was 8,400 with 68 per cent polymorphonuclear neutrophils. The Wassermann was negative. A flat plate of the abdomen revealed several fluid levels in the upper left quadrant in the dilated small intestines.

A diagnosis of intestinal obstruction was made and the abdomen explored. Approximately 2 feet of the jejunum were found to be gangrenous due to thrombosis of its arterial blood vessel. This was resected and bowel continuity re-established by means of an end to end anastomosis. The postoperative course was uneventful.

SUMMARY

An apparently healthy individual with no history of previous operations or predisposing illnesses received two injections for uncomplicated internal hemorrhoids. The injections were given in the accepted fashion and no local symptoms developed. Five days after the second treatment infarction of a portion of the superior mesenteric artery occurred as demonstrated at the operating table.

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RUPTURE OF RECTOSIGMOID DURING SIGMOIDOSCOPY

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SIGMOIDOSCOPIC perforation of the bowel is not so uncommon as the few cases in the literature would seem to indicate. A mortality rate of 56 per cent has been reported in those patients not operated upon within one hour after rupture (Strömbeck). Any bowel, healthy or diseased, in any patient, conscious or unconscious, but especially past middle life, may be ruptured by any foreign body inserted into the rectum for any reason. The death of the patient in this case report appears to have been hastened by sigmoidoscopic rupture of the bowel because bleeding per rectum was the result of terminal and extensive cardiovascular disease. This serves to remind us of the ways in which such perforation might be avoided. In addition, the use of present day chemotherapeutic and antibiotic agents, as demonstrated in this case, might reduce the mortality in both those who are operated upon early, and those operated upon late or treated without laparotomy.

CASE REPORT

This is the story, in brief, of M. S. (Case 178728), a fifty-two year old white woman who was admitted on November 1, 1946 to the medical service of Dr. William Leaman at Philadelphia General Hospital for treatment of advanced hypertensive cardiovascular-renal disease. The prognosis was very poor.

On November 15, 1946 it was decided to take an x-ray examination of the chest, since, in addition to a pleural effusion, pulmonary infarction, hypertensive encephalopathy, oral hemorrhage and other signs of cardiorenal decompensation with edema, mild uremia, anemia and acidosis, this patient was also showing some evidence of a pneumonia and penicillin therapy was contemplated. As she was about to be lifted from her bed, it was noted that she was lying in a pool of blood which seemed to be coming from her vagina.

The gynecologic resident examined the patient and found a large clot in the vagina but no other pelvic pathologic symptoms. He felt that the bleeding was dysfunctional in type but that it might be part of her general cardiovascular disease or the result of a fundal carcinoma. He recommended dilatation and curettage if vitamin K, ergotrate and an icebag did not stop the bleeding.

These measures, in addition to plasma, failed to stop the hemorrhage and the fall in blood pressure. In the evening of the same day, therefore, the gynecologic resident performed a dilatation and curettage but found nothing to account for the bleeding. He placed a finger in the rectum and found a large clot of blood, whereupon he summoned the surgical resident who had done a few sigmoidoscopic examinations and the latter attempted to study the patient with the usual 25 cm. Montague sigmoidoscope which had been boiled but which was not sterile.

Being uncertain of his visible findings and having felt something "give" on introducing the sigmoidoscope, he asked the author to see the patient. When this reporter arrived in the operating room, the patient was on her back with her feet in stirrups and still under the effects of intravenous sodium pentothal anesthesia. The sigmoidoscope was in the rectosigmoid to a distance of about 14 cm. from the anus. Upon looking into the instrument one saw an elliptical opening, horizontally placed, on the right anterior wall about 1.5 cm. wide, through which had prolapsed approximately 2.5 cm. of glistening, pinkish white, serosa-covered small bowel with two fat tags attached to it. As the sigmoidoscope was withdrawn a little, a small quantity of pinkish, thin liquid gushed through the opening. Dark, thick blood could be seen coming down the lumen of an atonic sigmoid from above 14 cm. The mucosa was pale, edematous and wrinkled into folds. It seemed to invaginate itself into the sigmoidoscope. Above the anorectal line the rectal wall was heaped into thick, pale folds and small clots were visible, one of which was adherent to an

eroded area on the left just above the anorectal line and a small clot was in a vein beneath this area. No actual bleeding spot was encountered.

This patient had received no enemas either on the day of or the day previous to this examination.

Because of the patient's precarious cardiovascular-renal status and the presence of shock, immediate laparotomy was not done. It was hoped that the small bowel would become adherent to the ruptured sigmoid and that antibiotics would overcome infection. She was transferred to the surgical service of Doctor John Bower and placed in the Fowler position in an oxygen tent, given 500 cc. of whole blood; 200,000 units of penicillin intraperitoneally and 100,000 units intramuscularly every two hours, parenteral fluids and vitamin K (15 mg. daily); and rectal and upper intestinal Wangensteen drainage was instituted. The patient had been previously digitalized for her cardiac decompensation. She was also given Lugol's solution because a basal metabolic rate determined earlier had been plus 45 per cent.

On this schedule she developed no distention and had only slight rigidity and tenderness in the lower abdomen although no peristalsis was audible. Her temperature rose to 101.5°F. within the first few hours after the rupture occurred and then returned to practically normal until her demise. The pulse rate became accelerated and remained so. The blood pressure rose from 130/180 to 240/140 mm. of Mercury. The white count rose from 14,200 cells per cm. with 94 per cent polymorphonuclears two days before the rupture occurred, to 47,800 cells per cm. with 99 per cent polymorphonuclears the day following the accident. The red cell count was 3,500,000 per cm. with 9.5 Gm. hemoglobin. Serum chlorides approximated 515 mg. per cent, the blood urea nitrogen rose to 51 mg. per cent, the carbon dioxide combining power was approximately 41 mg. per cent and the urine output was diminished.

The patient continued to be weak, irrational and mentally dulled despite additional plasma, blood, fluids and penicillin.

Induration of the rectal wall was noted four days following rupture of the rectosigmoid.

The local surgical condition seemed to have responded to conservative treatment but the medical condition of the patient continued to become worse and she died on November 21, 1946, approximately six days after the perforation occurred.

The clinical causes of death were given as: (1) renal failure with uremia due to chronic glomerulonephritis, (2) perforated rectum, (3) pulmonary infarction, and (4) hypertensive cardiovascular disease.

At the surgical staff conference the patient's death was attributed to the patient's disease, to an error in diagnosis and to an error in technic.

The pathologic examination revealed the sigmoidal perforation to be closed by an adherent loop of ileum and an abscess, 4 cm. in diameter, was found in the pelvis. This yielded Bacillus coli, enterococci, Bacillus Welchii and Bacillus Friedländer on culture. Microscopically, the enteric vessels were thrombotic and the ileum was necrotic. Uncomplicated diverticuli were present.

Extensive vascular thromboses, embolism and infarction were encountered in the heart, lungs, spleen, liver and skin (which was also jaundiced), with the pelvic, left hepatic, portal, subclavian and cervical veins prominently involved. Hydrothorax, pericarditis, bronchopneumonia, adrenocortical lipoid depletion, patchy pancreatic necrosis, aortic and cerebral atherosclerosis and general passive congestion were additional findings. The heart was enlarged and contained a mural thrombus. The kidneys showed benign arteriolar nephrosclerosis. Traumatic cervicitis and endometritis were present. (The breasts were absent owing to surgical removal for tumors.)

The pathologic cause of death was given as: "Hypertensive cardiovascular-renal disease with myocardial failure; multiple venous thrombosis; perforation of the sigmoid with a pelvic abscess."

MALIGNANT MELANOMA (MELANOSARCOMA)

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THIS case is reported for four distinct reasons: (1) Because of the rarity of the condition; (2) as a plea for proper and painstaking examination which unfortunately is not always done; (3) to submit to microscopic examination every piece of tissue no matter how small or in significant it may appear; and (4) not to be a proctologic agnostic who says that proctology consists of piles and nothing else.

Primary melanoma of the rectum is such an extremely rare condition that each authentic case report adds to the sum total of our knowledge of the disease. An important observation is the fact that when more than one case is seen, the diagnosis is made sooner because of one's previous experience.

The name melanoma is derived from its color which is a cross between violet and blue and is due to the deposit of melanin which is essentially of metabolic origin.

Ewing defines a melanoma as a pigmentiferous tumor arising from a specific mesoblastic cell, the chromatophore, and possibly also from epithelial cells which have been modified by pigment production. His view is that these tumors have their origin in the mesoderm yet histologically they appear as epithelium that has taken on a pigmentary function. He further states that Virchow recognized both a sarcomatous and a carcinomatous melanoma, the former exhibiting a diffuse structure of spindle cells, the latter an alveolar structure; Ewing employed the term melanoma for the entire group.

As to their origin, different views are held by different men but it has become the consensus of opinion and pathologists at present are leaning toward the view that all melanotic tumors have their origin in the nerve endings such as the tactile corpuscles. They are exceedingly malignant, grow more rapidly than carcinoma and metastasize to any organ of the body, including heart and spleen, by way of the lymphatics and blood stream. Secondary rectal invasion is not infrequent, owing to metastases from the original growth in other pelvic organs.

The description of melanoma in man was made in 1806 by Laennec. However, melanoma of the rectum was first described in 1857 by Moore. Paneth, in 1883, gave the first complete case record of a melanotic tumor of the rectum. However, the first comprehensive paper on the subject was written by Chalier and Bonnet in 1912, who, in addition to one personal case, collected and reviewed from the literature sixty-four reports of tumors of the melanotic variety and estimated that they represented 66 per cent of all rectal sarcomas and that they formed from 2 to 3 per cent of all melanotic growths in the body.

In 1936, Linder and Wood reviewed the literature and found seventy-nine authenticated cases. Weeks reports one case of large spindle-cell sarcoma and states that a review of the literature revealed one-hundred cases of sarcoma of the rectum. He further says that in 200 cases of malignant disease of the rectum, carcinoma is found approximately 199 times and sarcoma is found once. Of the rectal sarcomas the melanotic type appears twice as frequently as other varieties. This statement is well borne out by Kallet and Saltzstein who reported seven cases of malignant disease of the rectum, three of which were melanomas and one case each of myosarcoma and lymphosarcoma.

Allen, in the transactions of the American Proctologic Society of 1931, reported a case of melanotic carcinoma of the anorectal region which, at first sight, somewhat resembled a thrombotic hemorrhoid, from which these tumors must be differentiated

because melanoma of the rectum always occurs low down, either in contact with the epidermal portion of the anal canal or close to it.

CASE REPORT

Mrs. M. C., a white female, born in Russia, eighty years of age, was referred to me October 16, 1941. Two brothers had died of carcinoma of the stomach(?). Otherwise her previous history was negative.

She complained of pain and bleeding which had increased so that she decided "to do something about her bleeding piles," as she put it. She accordingly consulted a physician who removed her piles in his office by the fractional method. For awhile she was fairly comfortable but soon her "piles" came back and the bleeding and pain were worse than before.

Upon examination there was a bilateral mass originating at the anal verge. This mass practically occluded the anal canal excepting the right anterior quadrant. The left half was violaceous in color and semihard to the touch, while the right half was hard and had a crater-like depression in the center. Digital examination showed the sphincter to be rather tonic and another mass, hard to the touch, 1 inch long by ½ inch wide was felt in the left anterior quadrant.

The 10 inch sigmoidoscope was passed easily full length; the rectum and sigmoid were empty; the mucous membrane was normal in appearance; no gross pathologic condition was observed.

The barium colon enema and gastrointestinal series were negative. (These were done elsewhere.)

Having seen one other case, I suspected melanoma. She was therefore sent to the hospital for biopsy which was done under local anesthesia. Specimens from various parts of the tumor were submitted to the pathologist whose report was as follows:

Sections show hyperplastic tumor tissue consisting of large polyhedral and spindle-shaped cells with cyanotic nuclei and considerable variation in cell size and shape. A few multinucleated cells are also seen. Other cells have vesicular nuclei with distinct nuclear structure. Most of the cells contain melanotic pigment. There is very little stroma of fibrous tissue.

Diagnosis: Malignant melanoma.

MUCINOUS CARCINOMA ASSOCIATED WITH FISTULAS OF LONG-STANDING*

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THE occurrence of anal fistulas in intimate association with cancer usually poses the question of their etiologic relationship. At times the fistula is clearly the result of the breakdown of the neoplasm. But is the malignancy ever a sequel to the fistula?

There has been considerable skepticism in this regard. Jones[12] believes that the fistula in such cases is always secondary to the growth. Ewing[7] agrees with Kraske, whom he quotes, that "there is no evidence to prove that cancer develops in tissues altered by . . . fistulae or cicatrices." Kaplan and Rubenfeld,[14] during fifteen years on the Radiation Therapy Service at Bellevue Hospital, failed to find a single case in which a fistula was listed as antecedent to carcinoma. In Smith's[28] experience, "a fistula so rarely degenerates into cancer that the occurrence deserves notice."

Nevertheless, perusal of the literature reveals records of at least fifty cases in which cancer presumably developed in previously benign fistulas[1,2,3,5,6,8,9,15,16,18,19,20,21,23,26,27] or in scars following operations for fistulas or abscesses.[6,13,17,22,26,27,31]

It is difficult, almost impossible, in any given case to make certain that the fistula antedated the cancer. No matter how innocent its appearance, there is always the possibility that histologically the fistula may be harboring neoplastic elements. Even a negative biopsy report may be misleading.[5,6] The presence of tuberculosis,[21,31] lues[30] or lymphogranuloma venereum[5,16] may obscure a co-existing carcinoma. In addition, cancer may be so incredibly slow-growing as not to become evident for years.

Exhaustive histologic studies by Lynch and Gross[18] and Fitchet,[8] in an effort to establish a causal relationship between fistulas and associated carcinoma, failed to give convincing evidence.

Though proof be unobtainable, where a fistula has been present over an interval sufficiently long to rule out the preexistence of even the slowest-growing cancer, it seems reasonable to assume that malignancy supervened and was perhaps induced by the chronic inflammatory process. Such an interval, of course, varies; but set arbitrarily at ten years, it should be more than sufficient to eliminate any doubtful cases.

TABLE 1
MALIGNANCY ASSOCIATED WITH FISTULAS OF MORE THAN TEN YEARS

Authors	Duration of Fistula, (years)	Type of Neoplasm
David and Loring[5]	10	
	13	
Ducassi and Smith[6]	17	Colloid
	16	Colloid
Fitchet[8]	20	Squamous
Gabriel[9]	37	Squamous
Lisa[16]	10	Adenocarcinoma and squamous
Lynch and Gross[18]	15	Colloid
Meland[20]	10	Squamous
Moon[22]	10	Adenocarcinoma
Rosser[26]	15	Adenocarcinoma
	15	Adenocarcinoma
	10	Colloid(?)
Yeomans[31]	12	
	11	Colloid

Reviewing the fifty cases mentioned, in which fistulas were inferably forerunners of carcinoma, we find that only fourteen qual-

* From the Surgical Service of The Brooklyn Hospital, Dr. William H. Field, Chief Attending.

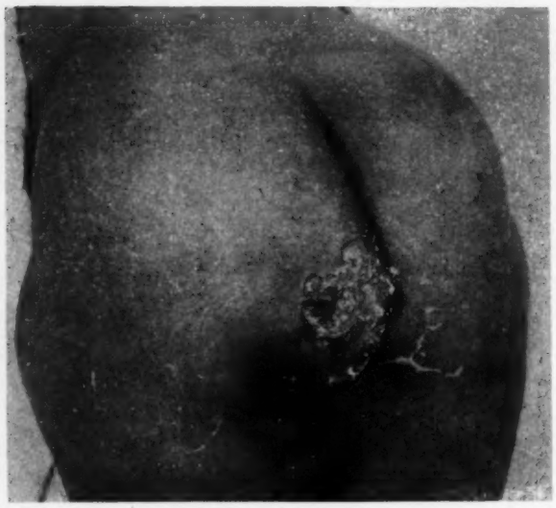


Fig. 1. Case 1.

ify under the ten-year interval limitation. (Table 1.) In addition to these three of our cases fall in this category.

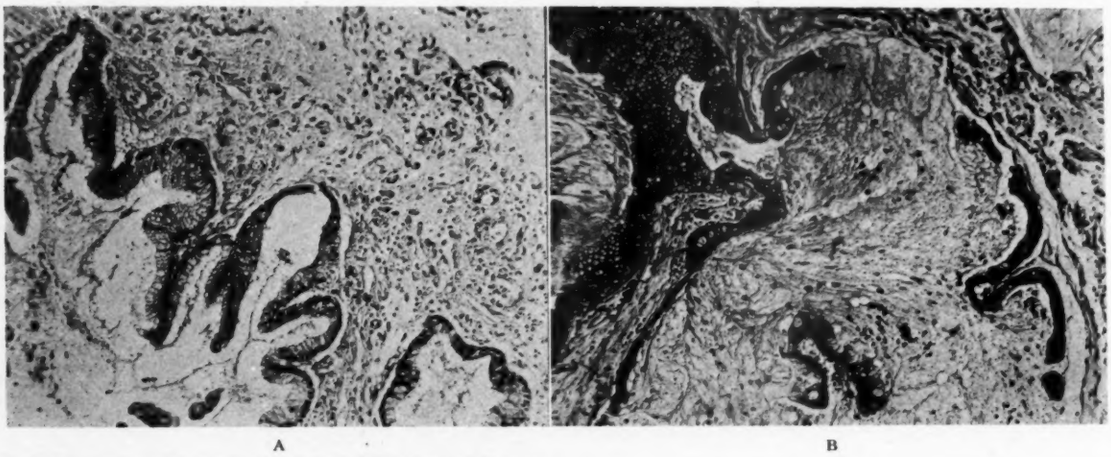
CASE REPORTS

Case 1. E. L., a sixty-one year old negro widow, was admitted to the service of Dr. Marino December 21, 1933, complaining of a discharging swelling of the left buttock. She had had an intermittently discharging fistula ten years, but during the last year the discharge had become almost continuous and a progressively increasing swelling about the fistula had developed.

On examination, there was a foul, cauliflower, ulcerated, non-tender mass, 6 inches in diameter, immediately to the left of the anal orifice, pierced by numerous openings, discharging mucopurulent material. (Fig. 1.) Two of these sinuses led to the ischiorectal fossa. In the left groin there was an oval, firm mass, 2 inches long. The blood Wassermann was four plus, but the Frei test proved negative.

A biopsy specimen from the perianal mass showed mucinous carcinoma. Figures 2A, B and C have been arranged by Dr. deVeer, Proctologist to the Brooklyn Hospital, to show, fields of progressively increasing pressure from the excess of mucus secreted by the tumor cells.

The enlarged inguinal node was removed and a few days later the tumor proper. It



A B

C

Fig. 2. Case 1. A, the coarsely granular structure of the columnar epithelium is suggestive of rectal mucosa. Mucus is present in abundance within the cells and distends the gland lumina. B, cystic gland spaces in the tumor have ruptured so that mucus has been permitted to escape. The fragmentary remains of epithelium show shorter columnar cells with less secretory activity, evidently because of pressure atrophy. C, in this field abundant mucus has dissected connective tissue but no tumor cells are present. At this site the secretory tumor cells may have atrophied and disappeared, or the mucus may have been formed in some neighboring focus.

was found to be intimately attached to the lowermost part of the rectum. The wound filled in in two months. Six years later a recurrent growth was excised from the left groin and the following year another from the thigh just below the previous growth.

When I saw her in June, 1947, six years since the last operation and thirteen since the first, she was evidently in good health with no signs of recurrence.

Case II. Mrs. A. D., a widow, aged sixty-four, was seen at her home in 1941 in consultation during an attack of pneumonia. Her daughter had noticed a perianal mass while inserting a rectal thermometer.

The patient related that she had had a fistula on the left side thirty years. During the last two years there had been an increasing, sticky discharge and a "hardening" of her left buttock.

The inner half of the buttock was infiltrated by a rigid mass, perforated by innumerable pinhead to pinpoint-size openings which presented droplets of glary, turbid fluid. One of these was found to be connected with a tract extending to the ischiorectal fossa.

During the remaining six years of her life she stubbornly refused to visit my office or to enter a hospital. I was kept informed, however, through her family physician. The growth inexorably progressed in size, ulceration and foul discharge. The patient gradually deteriorated and died of "pulmonary edema."

Case III. M. C., a seventy-seven year old colored widow, was admitted to Dr. Marino's service March 21, 1945, complaining of fecal incontinence, perianal swelling and discharge.

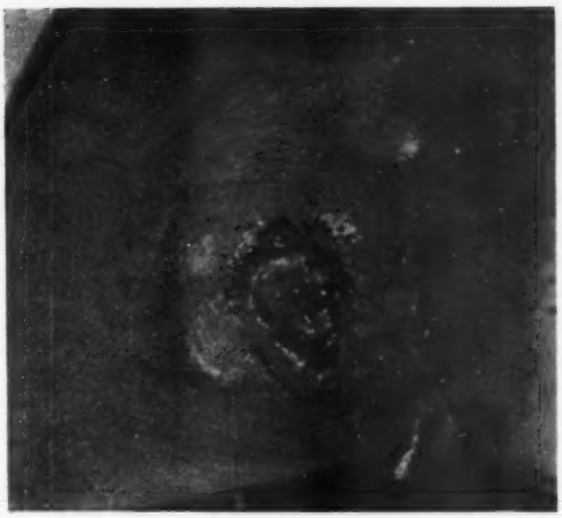
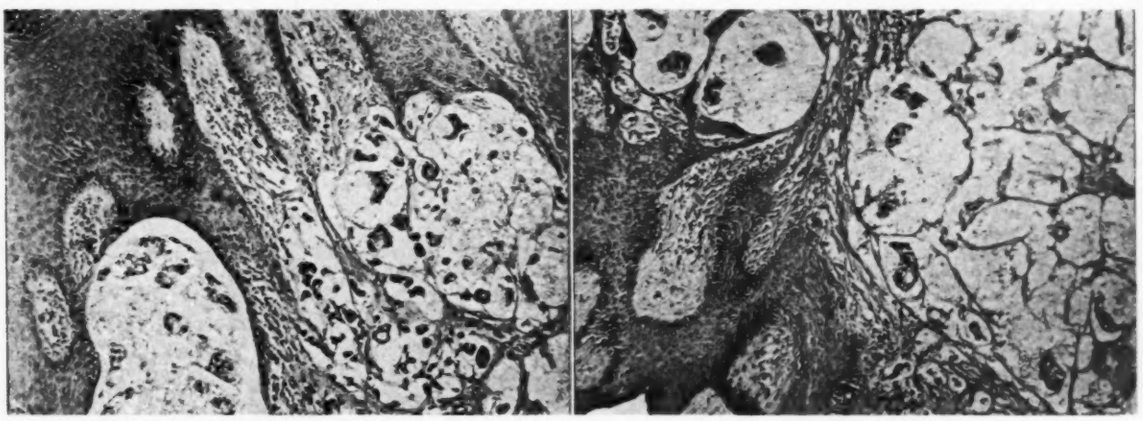


Fig. 3. Case III.

She had had intermittently a fistula or abscess to the left of the anal orifice fifty-five years, but during the preceding few months the pain, swelling and discharge had progressively worsened and her stools had gradually decreased in caliber to pencil-thinness; incontinence developed.

She was orthopneic and dyspneic on slight exertion. Her heart was moderately enlarged and fibrillating. The liver border was two fingers-breadth below the costal margin. She showed poor renal function.

Locally, a rigid, friable mass displaced the medial half of her left buttock and extended anteriorly to the left labium, which was elephantiac, and to the opposite side of the anal orifice. (Fig. 3.) It was honeycombed with numerous sinuses draining a glary, mucopuru-



A B

Fig. 4. Case III. A and B, the tumor structures consist largely of small glands and clusters of cells lying within masses of their own secretion. The mucus is seen dissecting its way up to and into the surface epidermis. These fields represent sites at which new fistulous openings were about to appear.

lent material. The involvement of the anus resulted in a stenosis which barely admitted the tip of the finger. In the left groin were small, firm, non-tender lymph nodes.

The blood Wassermann and Frei tests were negative. Because of absence of sensation in the tumor, a biopsy specimen was readily removed without the aid of an anesthetic. The pathologic diagnosis was mucinous carcinoma. (Figs. 4A and B.)

Because of the poor general condition of the patient, it was deemed inadvisable to risk surgery. However, she died two months later at another hospital of cardiac failure following a rectal resection.

COMMENT

These three cases bear a striking resemblance both in their histories and in the character of the growth. Recurrent abscesses and fistulas of many years' standing take on the aspect of an expanding, invasive neoplasm, begin to discharge an irritating mucous secretion in excessive quantities and ulcerate. These changes are gradual and progressive.

The question of the probable origin of this glandular type structure in the perianal region has intrigued many observers. It is believed that the answer is most likely to be found in the observation of Hermann[11] in regard to the presence of erratic glands, closed follicles and gland ducts connected with the anal crypts.

Besides exemplifying carcinogenic potentialities in old fistulas, these cases are of interest because mucinous or colloid carcinoma is very rarely seen in the anal region, particularly so in negroes. Of the fifty-one cases of anal carcinoma in the group reviewed by Buie and Brust[2] at the Mayo Clinic, only one was colloid in nature. Rosser[25] found only one of this type among thirteen anal malignancies in his experience. At the Brooklyn Hospital, Cases I and III were the only mucinous cancers among the twelve anal neoplasms in the past fifteen years.

It is especially remarkable that these two cases should have occurred in colored patients in a hospital predominantly white. At Johns Hopkins Hospital carcinomas in Caucasians outnumber those in negroes four to one. Terrel[29] has seen only one anal cancer among negroes in Richmond which is one third colored. Gant,[10] with large negro clinics, never saw or heard of rectal carcinoma in a negro. Rosser[25] saw five cases and Kraker[15] one.

SUMMARY

Of the fifty cases of fistulas on record in which malignancy evidently supervened, probably not all truly represent carcinomatous transformations of previously benign fistulas. However, at least fourteen gave a history of a fistula of more than ten years' duration, long enough to rule out antecedent malignancy.

In addition, three of our cases are reported in which fistulas of ten, thirty and fifty-five years' duration, respectively, developed carcinomatous changes. These growths were all of the mucinous type, two of them in negroes.

CONCLUSIONS

Chronicity of a fistula should make us aware of the possibility of malignant change.

Whether or not the reported cases really represent cancer formation in previously benign fistulas, it is safest to eradicate fistulous tracts as early and as completely as possible.

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Papers of the Scientific Session

LEIOMYOMA WITHIN THE SUBSTANCE OF THE SPHINCTER

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SMOOTH muscle tumors of the gastrointestinal tract are of frequent occurrence in the upper segments but few have been reported involving the colon, fewer still the rectum and I can find no report of occurrence as distal as the lesion which I shall describe. Leiomyoma may be malignant or benign and is often sufficiently involved with fibrous elements to make the term fibromyoma more accurately descriptive.

The best resume of the problem that we could find in the literature was a paper in Surgery, Gynecology and Obstetrics December, 1941, by Thedore Golden and Arthur Purdy Stout. They review the problem for the entire gastrointestinal tract and report one sigmoidal lesion which was malignant and one of the descending colon which was benign. Five rectal cases are reported: one of the lower anterior wall, one malignant tumor of the lower posterior wall, one not described, one pedunculated tumor of the mid-lateral wall and one

Fig. 1. Leiomyoma of anal sphincter.

of the lower anterior wall removed, incidentally, in the course of perineal prostatectomy.

C. Allen Good, in Radiology of December, 1942, reports a large myoma of the distal portion of the transverse colon. This lesion was diagnosed by roentgen study, preoperatively.

William Lawrence Estes, in Annals of Surgery, Vol. XLIV, reports large myofibroma of the sigmoid. Diagnosis in this case was clouded by pathologists' suggestion of tuberculoma.

A bibliography of a report of a fibroma of the ileum by John W. Dewis, Boston Medical and Surgical Journal, October 18, 1906, includes four retrorectal tumors reported by four foreign writers.

H. H. Varner and J. L. Green Jr., in Southwest Medicine, 1935, Vol. 19, describe a small myoma of the sigmoid which was resected and primary anastomosis done.

CASE REPORT

R. P., a middle-aged female, was admitted to The Milwaukee County General Hospital on October 7, 1946 for treatment of purpura. The purpura responded satisfactorily to medical management and proctologist's consultation was requested on December 16, 1946 because of bleeding at stool and pain at the anus.

Examination revealed a firm, tender mass posterior to the anal canal. No fluctuation could be noted but surgery was advised for what was thought to be a small, chronic, postanal sinus or abscess. Upon opening the overlying skin it became obvious that the mass was a solid tumor. It was sharply demarcated and was easily dissected free from the normal sphincter muscle in which it lay. The tumor, about 3.5 cm. by 2 cm. in size, was of greyish red color and friable. The wound was left open to heal by third intention.

The pathologist's report was: "Leiomyoma, histologically benign."

It seems that the origin of this tumor must have been either the terminal portion of the longitudinal muscle or the fibers of the internal sphincter. Recheck examination on January 29, 1947 showed the wound to be well healed with no evidence of residual mass.

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PRIMARY POSTOPERATIVE HEMOSTATIC PROPHYLACTIC DRESSING IN ANORECTAL SURGERY

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THE demand for a prophylactic and hemostatic primary postoperative dressing in anorectal surgery has long been recognized. One that is less painful, non-irritating to tissue and not too bulky; one that is absorbable or easy to remove and simple in application; one that is adjustable to slight or tight, constant pressure to fit the need in the individual patient and one that will permit early expelling of gas and not interfere with catheterization or urination.

A new material in the form of oxidized cellulose gauze* used in conjunction with the sulfanomides has aided much in making such a dressing. The dressing consists of: (1) A thick strip of domestic or gauze to be used as a belt around the waist. (2) A large, perineal pad made of a piece of cotton ½ inch thick, 4 inches wide and 12 inches long, placed and folded in the center of a piece of gauze 28 inches long. This makes a pad 12 inches long with 8 inches of gauze on each end to be pinned to the bandage around the waist. (3) 5 Gm. of sulfathiazole or sulfanilamide powder. (4) One piece of 3 by 3 inch oxidized cellulose gauze. (5) One curved Kelly artery forceps to insert the oxidized cellulose gauze. (6) Two to four pieces of 4 by 4 inch sterile gauze pads. (7) Three large safety pins and (8) Two pieces of adhesive 2 inches wide and about 20 inches long.

APPLICATION OF DRESSING

The application of this dressing is most important. At the completion of the operation with the patient in a jack knife posture, 3 to 5 Gm. of sulfathiazole or sulfanilamide powder is dusted and rubbed into the anal and rectal wounds with the gloved finger, care being taken that it is smoothly distributed and not left in piles to act later as foreign bodies in the wound.

Fold a piece of 3 by 3 inch oxidized cellulose gauze to match opposite corners thus making a right angle triangle. With a pair of curved Kelly artery forceps, grasp the folded gauze along a short side of the triangle so that the end of the forceps will be at a point of the long side. Insert the gauze with the forceps about 2 to 2½ inches into the anal canal so that it fits the slit-like shape of the anus and will come in contact with all the cut surfaces of the anus and lower rectum. This leaves half the gauze to be placed in or over the cut surfaces outside of the anus. Carefully remove the forceps so as not to displace the inserted oxidized cellulose gauze.

Place two to four pieces of 4 by 4 sterile gauze pads over the oxidized cellulose pack to fill in the space between the buttocks. Change the posture of the patient from a jack knife position to a ventral prone posture by flattening the table. Tie or pin the gauze band around the waist. Place the perineal pad over the sterile gauze pads and pin one end of the gauze, in the back, to the band around the waist. The patient is raised slightly to one side and the other end of the perineal pad brought forward, pulled tightly and fixed securely by pinning it in front to the bandage around the waist so as to make an upward and forward pressure on the anal dressing.

The dressing is completed by applying two strips of adhesive, 2 inches wide and about 20 inches long, strapped across the buttocks over the perineal pad. In people who have lost much weight or have relaxed redundant skin folds and poor muscular tone, the adhesive must extend well around

* Distributed by Parke Davis & Company under the trade name "oxycel.")

on the outer side of both hips to make constant pressure on the anal region.

This dressing is especially useful after hemorrhoidectomy for internal or combined hemorrhoids, anal, anorectal or rectal fistula, polyps, new growths or any other operation within the anus and lower part of rectum.

OFFICE DRESSING

As a dressing to be used in minor procedures in the office, such as excised thrombosed external hemorrhoids, skin, tabs, small anorectal polyps, anal warts, subcutaneous fistula, simple fissure and abscess, a strip of oxidized cellulose gauze placed or packed into the wound is usually sufficient dressing to control bleeding from the incised surface. A much safer and probably just as comfortable dressing is made if fixation and constant pressure is added. The shape and size of the oxidized cellulose gauze and the amount of pressure and fixation to be applied depends on the amount of bleeding and the size and shape of the wound to be dressed. As a rule the dressing can be removed in twenty-four hours without pain or bleeding.

VALUE AND USE OF OXIDIZED CELLULOSE GAUZE

Sufficient research and clinical use of oxidized cellulose gauze have proved that it is non-irritating to tissue and it has definite but limited hemostatic properties. Its value must be classed as only an adjunct to other methods of hemostasis.

Oxidized cellulose gauze applied directly to an oozing surface tends to adhere, a coagulum is formed and bleeding stops. When bleeding is brisk, the blood tends to push or wash off the oxidized gauze and prevent clotting. I have not found, as suggested by Rosser in the Southern Medical Journal, December, 1946, that small pieces of the material applied to each wound is sufficiently safe to be depended on as a hemostatic dressing. Certainly if bleeding is free, fixation of dressing and pressure should be added. If a spurting vessel is present, ligature is indicated and then the dressing applied.

Oxidized cellulose gauze when dry is pliable and easy to insert or apply to a bleeding surface. When the gauze becomes saturated with blood, it swells, turns dark brown and controls bleeding from the oozing surface. Within ten to twenty-four hours a film of gelatinous substance is formed around the gauze and the gauze turns dark brown and becomes friable or jelly-like depending on the degree of saturation with blood or fluid of the body tissue. This film prevents granulation from becoming involved into the mesh of the gauze, makes separation from tissue easy and, if pressure is not too tight, permits the early expelling of gas around the pack.

At the end of six to eight hours the strips of adhesive may be partly removed to see if blistering from the adhesive has occurred. If not, the dressing and packing is removed the next day. If the bleeding is sufficient to make complete saturation of the gauze, the gauze at the end of twenty-four hours is very dark brown in appearance and so friable that it may be easily pulled off in small pieces. However, in most cases, using gentle manipulation, the gauze can be separated from the tissue without discomfort to the patient. There is usually no bleeding in removing the gauze pack and the tissue is left dry, with little if any edema.

COMMENTS

After the dressing and pack is removed the wound is dusted with sulfonamide powder and two 4 by 4 sterile gauze pads are placed over the wound and fixed in place with short, narrow strips of adhesive. Hot fomentations are started in the afternoon of the second day.

The dressing should be applied quickly after the rubbing of the sulfonamide into the tissue and the mopping of the blood out of the wound to prevent further bleeding before the dressing is complete.

Hemostasis in this dressing is controlled first by the natural clotting of the blood second, by the hemostatic properties of the

oxidized cellulose gauze and third, by the constant pressure of the gauze against the bleeding surfaces to prevent displacement or expelling of the pack in vomiting, coughing, sneezing or exertion, giving time for clotting and the formation of thrombi in the open end of the vessels.

In the female, when cathterization or urination is necessary, the front portion of the pad may be unpinned and pulled backward out of the way leaving the rectal dressing secure and in tact. After the procedure is completed the pad is repinned in front.

I have used this dressing in more than 400 hospital cases. In no case has bleeding been sufficient to require a change or reinsertion of the packing.

When there is no evidence of bleeding on the bandage, pressure from the strapping of the buttocks may be released in from six to eight hours. When there is some evidence of bleeding on the dressing, pressure should be continued for twenty-four hours to give time for firm clotting in the vessels.

Beside the hemostatic effect of the oxidized cellulose gauze, the firm incompressible structure of the gauze makes it especially suitable for a dressing when pressure from the packing may be needed to control bleeding in the anus and lower part of the rectum, following hemorrhoidectomy, cryptectomy, fistulectomy, anorectal polyps, tumors, proctotomy and so forth.

There have been sufficient experimental investigation and clinical use of the oxidized cellulose gauze to prove its specific hemostatic properties. It has little value when applied to a spurting arterial vessel as the pressure from the bleeding vessel is sufficient to push off or wash away the clot. Therefore the spurting arterial vessels should be ligated and dependence should not be put on the hemostatic effect of the gauze to control this type of bleeding.

The perineal pad is made and wrapped in individual packages and sterilized along with other dressings in surgery. The chief objection that I have found to this dressing is that the adhesive strapping across the buttocks will occasionally cause blisters. This may be prevented by the early loosening (ten to twelve hours) or removal of the adhesive.

SUMMARY

1. A prophylactic hemostatic primary anorectal dressing has been described.

2. The method of application of this dressing is most important.

3. Sulfathiazole or sulfanilamide powder rubbed into the wound at the time of operation is the prophylactic portion of the dressing.

4. Oxidized cellulose gauze has specific hemostatic properties sufficient to control hemorrhage when applied directly to an oozing surface; when bleeding is free, definite fixation of dressing and constant pressure should be added. If a free spurter is present, a ligature should be applied.

5. Oxidized cellulose gauze saturated with blood turns dark brown and becomes friable. A thin, moist film is formed around the gauze within twenty-four hours, making removal easy without starting of bleeding. The tissue is left dry with little or no edema.

6. The thin waist film or coat of coagulum around the oxidized cellulose gauze pack permits early expelling of gas around the dressing.

7. An oxidized cellulose gauze pack if left in the tissue is absorbable.

DISCUSSION

Harry W. Christianson, (Minneapolis, Minn.): Dr. Pruitt has presented a very timely paper on the use of a relatively new postoperative hemostatic agent. He has described his postoperative dressings in very minute details.

We may not all agree with his technic of postoperative dressings but we can rest assured that his method accomplishes the end result.

I do not agree with Dr. Pruitt in the application of oxycel in every operative patient but we cannot deny the fact that this oxidized cellulose gauze or similar hemostatic agent is a very valuable adjunct to our armamentarium in preventing and treating postoperative hemorrhages.

Dr. Pruitt wisely admonishes against the use of oxycel when arterial bleeding is present.

Suturing then is absolutely indicated if the surgeon wishes to sleep that night.

We use oxidized cellulose gauze or similar preparations postoperatively, only when generalized venous oozing is present. This avoids the use of excessive suturing and hence reduces the postoperative pain as every suture is a potential producer of pain in the anoderm and perianal skin.

The destruction of polyps in the rectum or sigmoid by fulguration in my experience has never indicated the need of any hemostatic agent as fulguration is sufficient. However, if we fulgurate a large, sessile polypoid mass with or without malignant degeneration then oxycel is placed over the area and held in place with packs of gauze.

When the need for oxidized cellulose gauze is indicated, we usually insert this hemostatic agent in the rectum in the form of a cylindrical roll until 1 inch or less protrudes from the anus. The protruding exterior portion of this "oxycel" pack is then split longitudinally and reflected laterally to cover any raw surface external to the anal margin. Four by 4 inch gauze sponges are then packed in between the separated ends to fill in the space between the buttocks. This places the hemostatic agent against the raw surfaces after which an adhesive strap is applied tightly for pressure. Three to four hours postoperatively, the dressings are removed but the oxidized cellulose gauze is left intact. The oxycel is removed in forty-eight hours or, if deemed advisable, it is left in place and expelled later with the first bowel movement.

We do not recommend the routine application of sulfathiazole or sulfanilamide to anal and rectal wounds; however, in grossly infected patients its use may be indicated.

Before the advent of these hemostatic agents such as oxycel, thrombin topical and gelfoam we sometimes had to resort to large gauze packs to control postoperative bleeding. The removal of these packs was very painful and frequently required the use of an anesthetic. Now in its stead we use one of the hemostatic agents, preferably "oxycel." The removal of these packs is painless; in fact, it is usually unnecessary to remove them. Dr. Buie would say, "why worry, the current is always to the south."

In conclusion, I want to commend Dr. Pruitt on his usual fine presentation of a paper and on his observations on the effect of a new and valuable aid to postoperative hemostatis.

Dr. Jack Kerr (Dallas, Tex.): Oxycel gauze is the outgrowth of the search for agents that would carry the administration or the application of thrombin, thrombin, of course being a very powerful coagulant. This search for a material to be absorbable and yet non-irritating has been going on for some while, the thought perhaps being advanced many years ago, even by Cushing, when various experimental studies were carried on with fibrin itself. The first of these preparations was fibrin foam, which later included oxidized cellulose, both cotton and gauze, and the gelatin sponge and starch sponge.

As it is marketed—oxidized celluloses, both the cotton and the gauze—if thrombin is to be used topically with the application, alkalinization becomes necessary. This, of course, is done by the addition of a 1 per cent sodium bicarbonate solution to the gauze after which it becomes very friable.

In later studies, it was found that these oxidized cellulose preparations alone had certain hemostatic properties, perhaps not as good as thrombin itself, but definitely present. They are also described largely on the basis of a styptic action but apparently there is some chemical reaction between the cellulosic acid, which oxidized cellulose is, and the salts of hemoglobin.

Our experience with these preparations includes three: the oxidized gauze and cellulose and the gelatin sponge. Our personal preference for these three is the gelatin sponge soaked in thrombin. It is a little more easily applied and, of course, including the thrombin we believe that the hemostatic properties of this is superior to the oxidized cellulose.

I think Dr. Pruitt is eminently correct in emphasizing that these types of dressings are not hemostatic to the extent that they exclude the necessity for ligature of any definite bleeding that is occurring at the close of surgery. A generalized ooze, as Dr. Christianson mentioned, is a definite indication for these preparations; they help a great deal. I do believe some amount of pressure is best. It does decrease the amount of suturing that is necessary and, therefore, cuts down the amount of postoperative spasm and pain that results following anesthesia.

We do not believe that these preparations should ever be used to the exclusion of the ligature method as Dr. Pruitt mentioned.

I enjoyed Dr. Pruitt's paper very much.

RELATION OF FUNCTIONAL TO ORGANIC DISEASES OF THE ANUS, RECTUM AND SIGMOID COLON

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DURING the past five years there has been a notable impetus given to the work of Freud and his associates in their studies on the relationship of the psychic behavior of individuals toward disease. This has been largely due to the influence of the war years, during which time great opportunity was offered in the mass study of the great number of men who were selected or rejected for service in the armed forces. The strain of combat also yielded excellent opportunity for the psychiatric study of a large number of men under abnormal conditions. The fortunate part of the entire study was the fact that strenuous efforts were made in standardizing psychiatric diagnoses and evaluating them on a scientific scale.

It must be realized that here we are dealing with something intangible. Bold and often strange theories may be presented which can only be given consideration as such until improved physiologic methods are devised to aid in final evaluation of the mechanisms responsible for functional disease. In this presentation, it is not possible to discuss the details of this work as the ramifications are considerable and it is a field in which special training is necessary for good exposition of its use, value and correct approach. However, there are many occasions in common daily practice in which the knowledge of the principles of psychosomatic therapy can be used to advantage. I believe most of us have the feeling that when psychosomatic problems are discussed, the concept becomes one of exclusion of organic disease. If no organic disease is found, then it too often is considered a functional problem and the patient is labeled a neurotic. At this stage the condition is treated symptomatically without any further attempts to inquire into the background or offer any help to the patient from a treatment standpoint of his functional disease. The problem should be evaluated briefly and if found to be deeply seated, requiring much time and possibly specialized knowledge, then referral to a psychiatrist is advisable. Not only is it possible for both functional and organic disease to be present at the same time but its combination exists in practically all diseases of the anus, rectum and sigmoid in varying degree. It must follow then that we become interested in the *entire* problem. Weiss[1] stated, "Psychosomatic medicine does not mean that the soma should be studied less but the psyche should be studied more."

Most psychosomatic diseases are based on the existence of a vicious cycle consisting of psychic and somatic factors and, depending upon whether or not it is the psychic or somatic component that is cured, the cycle may be broken. Therefore, a psychoneurosis may run an acute course and recover spontaneously when the basic somatic disease disappears, providing this disease is the precipitating factor. It is also possible that some unrecognized organic disease may very often stimulate a neurosis or cause a neurotic condition.

I am sure that you have all made the observation that most patients visiting a proctologist, at least for the first visit, are usually frightened, nervous and anxious. This is especially true of the female patients and, therefore, great care must be taken to put the patient as much at ease as possible. The first meeting of a patient with a physician can be one of confidence, trust or dependence; or it can be one of distrust, caution or defiance. The psychiatrist

states that transference neuroses may immediately be founded at this meeting. For instance, in the background of the patient there may have been a childhood relation of distrust toward the parent. This distrust or anxiety may be transferred to the physician and a neurotic attitude develops. This fact is important when the referring physician states, "I have examined this patient thoroughly. I am sure she is a functional problem, but I am unable to convince her that she does not have organic disease. I want *you* to handle this problem."

A more detailed history is important in the study of functional proctologic problems. A general rule that might be followed in the selection of a neurotic is that they are usually overactive and oversensitive. They may object or even refuse to give details of their childhood or their relationship with their parents or close relatives. In these individuals it is important to deal more thoroughly with a short analysis of the problem as it is presented by the patient.

During the examination procedure draping, comfortable position and gentleness must be given its proper attention. A physician with a reputation of kindness and gentleness will always be greatly appreciated. During the examination it is well to continue conversation with the patient in order to divert him from fear of the examining procedure. One of the greatest anxieties of the majority of patients is that a malignancy is present. If following the examination no malignancy is suspected or found, it is important that the patient be told this fact immediately and in an assuring tone. This follows the psychotherapeutic principle that when neurotic symptoms are divorced from the fear of organic disease, cancer for example, it loses the force, whereupon the slogan "carry on in spite of symptoms" often helps the patient a great deal.

One must face a psychic problem when recommending hospitalization or surgical procedure. Many patients have a great fear of any type of operation no matter how minor and even the thought of being admitted to a hospital is a great shock to them. It is, therefore, important that this be done in a manner which does not impart a great impact. This can be done by appealing to common sense values. A word might be added as to the worth of a secretary or nurse who is courteous, polite and is taught these same principles.

Much has been written and attributed to the physiologic act of defecation. Psychiatrists are very prone to believe that many of the mental disorders in adults and children can be attributed to abnormal habits concerned with this function. Whether this premise is correct or how frequently it occurs is not the purpose of this presentation. However, many psychologic problems are discovered to be the source of diagnosis in children of fissure-in-ano or poor bowel habits who are often referred by a pediatrician. The organic pathologic condition is preceeded by poor bowel training in many instances and often a definite bowel complex is already present in these young children.

The matter of bowel function in children and its relation to the psyche can be explained as part of their normal development. The first year or two of a child's development is the oral stage and the following two years can be considered the anal stage. Here children find that they have control of the sphincter and there is pleasure in its realization. Instead of it being a natural function and consumated when desired, it is, at an early age, definitely molded to fit the needs of our mode of living by having a movement at approximately the same time each day in an unnatural receptacle and position. The child will give up the newly found control if a pleasurable state can be substituted such as love for the parent and the surroundings. This is usually sufficient compensation for the average child. Difficulty in bowel training arises very often because of the exaggeration of its importance by the parent or overaccentuation of the basic return by the child as compensation

for the giving up of the pleasurable act. If toilet training is begun too early in life, the child may feel punished and think he should never have a defecation. The bowel habit then becomes irregular, a laxative is given by the parent the child refuses the laxative because he knows what the laxative will accomplish and believes it is not the right thing to do. In adult life it is the extremely meticulous housewife type that shows a carry over of this condition. It often becomes a compulsive state which is very difficult to overcome. In the treatment of functional types of constipation and diarrhea the proctologist will often find emotional factors which are the basis of the dysfunction. This is true not only in adults but in children as well. The factors involved have been already referred to but it may be stated that fear and anxiety increase the secretory and motor functions of the colon producing diarrhea while depression and despondency decrease these functions and produce constipation. Von Bergmann and Lenz[8] have demonstrated a mass peristaltic movement which occurs one or twice daily, consisting of the sudden relaxation of a physiologic sphincter located near the distal portion of the transverse colon with a simultaneous contraction of the cecum and ascending colon. If this fails to occur, it can be due to either atonia because of the lack of propulsive force or blocked by spasm in the spastic type. With this in mind a proctologist can often recognize these simple principles and clear up a situation which is most important to the patient and the parent.

A little psychotherapy will go a long way in postoperative treatment which may produce a painful response. Talk with the patient in order to divert his mind from anxiety.

Mucous colitis or irritable colon is probably the most common proctologic ailment which is largely of a functional nature and can be greatly improved by psychotherapeutic treatment. Because of this relationship, the condition has received a great amount of attention by the psychosomatic school. White, Cobb and Jones[2] regard mucous colitis as a bodily reaction rather than an organic disease. It is apparent that the autonomic nervous system plays an important rôle in the syndrome of irritable colon and that this dysfunction can be brought about by emotional factors. In general, it might be stated that stimulation of the parasympathetic system produces increased muscular tone and peristalsis with relaxation of the sphincters. Stimulation of the sympathetic system produces relaxation of muscular tone and diminishes peristalsis with spasm of the sphincters. It is possible to furnish evidence that there is active instability of the autonomic nervous system in persons with mucous colitis. This can be established by making use of various physical efficiency tests, among which there are the tests of Schneider[3] and Turner.[4]

There appears to be a definite type of patient who exhibit the intestinal findings of mucous colitis. They have been divided into three groups according to Alexander, Kahn and Adler: The first type, or the upper gastrointestinal type which exhibits cardiospasm and peptic ulcers, are usually active, independent, efficient and show overcompensation for inferiority. The second group, or lower gastrointestinal type, usually show symptoms of diarrhea and are definitely of the inferior type who are dependent, have a sense of guilt and a need for giving. The third type, also lower gastrointestinal usually showing constipation, are the vain individuals, having no sense of guilt or a need for giving and depend upon prestige for their receiving. The collection of these three groups with emotional makeup so expressed might give a clue to the proctologist in the recognition of an undetermined basic psyche. Jordon[5] has stated that we are accustomed to think of functional gastrointestinal diseases in two categories: (1) that which is psychosomatic, altogether or in part and (2) that which is somatic in origin. It is her opinion that symptoms of short duration are often on a psychogenic basis and in these instances

psychotherapy is completely effective. In the group that show symptoms of longer duration even though psyogenic in origin, it is necessary to treat the somatic condition. In the second group of patients she believes functional improvement is on a somatic basis.

A word concerning the radiologic evidence of mucous colitis might be important at this point. With the ordinary preparation used by most radiologists for a roentgenologic study of the colon, spasm and irritability are set up even in normal individuals by the laxative or enema used in the preparation. This will make the study valueless insofar as the diagnosis of mucous colitis. The ordinary preparation should then be considered as a search only for organic disease of the bowel. It becomes necessary to consider an additional study when one is looking for functional disturbances and in this instance x-ray should be performed without any preparation whatsoever.

In evaluation of the diagnosis of mucous colitis it is important that all tests be done first so that organic disease is fully eliminated before the diagnosis and treatment is given the patient. If the proctologist back-tracks later and asks for tests to rule out organic disease after having stated that only functional disease existed, all confidence toward the physician is lost by the patient. It should be explained that his symptoms are due to overexcitement of the bowel and that he should never use cathartics. A low residue diet and elimination of any irritating physical factors are important. The use of a synthetic atropine derivative is often helpful in the acute stages of the disorder. It is important to discover the cause of tension in each of these patients. If the correct insight is given, adjustment made in their activities and constant reassurance given, the emotional tension producing the attacks can be lessened.

The psychosomatic factors concerned in ulcerative colitis are only debatable insofar as the extent that the emotional background plays in the etiology and treatment of the disease. Murray and Sullivan[6] have stressed the importance of psychogenic factors in the etiology of ulcerative colitis and consider psychotherapy very important in its treatment. They believe that when the emotional conflict is solved and there is no longer this irritating factor, intestinal motility returns and the bowel can take care of any bacterial invaders so that the disease should promptly improve. The fear of the disease in itself, the fact that it occurs most frequently in young individuals who are often the sole means of family support will cause psychic factors which aggravate the patient's general condition and, in turn, the local disease. These factors, therefore, should be either eliminated or at least ameliorated. Bargen and Jordan,[7] in a symposium on the treatment of chronic ulcerative colitis read before this society in San Francisco last year, attested to the great importance of psychotherapy in the treatment of the disease. I believe that a practical view can be taken regardless of the actual etiology of the disease, whether it be organic or psychic. A certain varying degree of emotional back-ground can be found which has been present previous to the initial disease or immediately before a flare-up of the chronic form in almost every patient afflicted with the disease.

Pruritus ani also has its psychiatric elements. The real percentage of patients with pruritus who have a functional background cannot be stated. Regardless of this, I believe it is important for us to know that in any of the patients, even when it may be entirely of organic etiology, there is most often a varying proportion of mixed psychic disturbances and organic elements. Stokes[9] has cited the case of an elderly gentleman who refused treatment of his ringworm with the frank remark that the disease and its attendant scratching resulted in more pleasant sensation than sexual relations with his wife. This instance represents the masturbatory element that is associated in patients with

pruritus and shows the masochistic element inherent in the concomitant suffering and pain. These patients are usually overactive and oversensitive. Overactivity results in exhaustion and oversensitivity produces exaggerated emotional changes which increase the exhaustion. The fatigue does not stimulate the patient to the ordinary tired feeling but stimulates abnormal reflexes and causes lack of inhibitions. For example, increased sweating in an overactive patient with a definite fungus infection results in more optimal growth requirements and might explain a flare-up of fungus infection.

CONCLUSIONS

It has been the aim of this presentation to show briefly that psychic disturbances of varying degree are present in the majority of proctologic disorders. If should be recognized that since organic pathology may produce psychic disturbances and functional disease can produce organic disease it follows that they are usually present simultaneously. It is important that the psychic component be evaluated and, if necessary, treated in the light of our present concepts of psychotherapy.

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DISCUSSION

Richard H. Appel (Indianapolis, Ind.): I find Dr. Schneider and his paper to be much like a troublesome conscience that has come back to plague about a personal weakness which is a dislike of the treatment of psychopathic patients. As a result, my efforts in the past have been directed chiefly towards a diagnostic end. So to a certain degree, I represent a negative side in this discussion. I am sure that one of the reasons which led me to enter the field of proctology was the idea that the pathologic conditions encountered were both visible and palpable and it is with this attitude that most of our competitors in the general surgical group begin and end. I have learned that this is not true, that often the task of separating somatic from the psychic presents one of the most difficult of diagnostic problems. It is here that our knowledge and judgment attain their highest value because we are often called upon to be the final judges in this question. This group of patients is the private preserve of the proctologist whether he likes it or not. There is no overlapping field for the two branches of proctology and psychiatry and often patients pass each other between the two offices. Particularly in adults, however, more are apt to arrive in proctologists' offices than to depart.

I find that I am more comfortable in the presence of the psychiatrist since the war than before. Perhaps that is because I saw more of them in the service, lived with them and may even have been somewhat inoculated with some of their theories but I do not believe so. I learned to respect them but found that V-J day cured more of their patients than any single therapy. I think they would be more grateful to us for proctologic therapy than for assimilation of their theory. I understand there is now considerable discussion among them as to the importance of organic dysfunction in contrast to those who are interested chiefly in functional disorders as a basis of the patient's trouble. Apparently there is no retreat from Freud but there is less emphasis. So our efforts, I believe, should be directed towards greater diligence in correcting minor defects. This is frequently

done at the instigation of the psychiatrist himself, however, with mixed results.

I do find myself looking more and more closely at the increased thickness of the rectal mucosa, very slight prolapse, increased sphincter spasm, etc., and mulling over the minor complaints.

W. A. H. Scheffler (Camden, N. J.): Research to measure and make perceptible to our senses and control, forces which were not visible or perceptible before, lately has been making great strides—stratosphere, cosmic rays, radar and nuclear fission are just a few examples.

It behooves medicine to make the same progress in the evaluation of processes which are not directly perceivable and measurable but rather intangible. I think Dr. Schneider's paper has contributed in a most excellent manner to this problem with his study of the psychosomatic responses and their relation to the gastrointestinal tract and I wish to congratulate him.

The few minutes available for discussion will not permit me to go into many details of this paper. I am therefore trying not to repeat anything that has been said and just elaborate on a few points of this fascinating subject. Dr. Schneider mentioned the anal phase of the child, during which toilet training takes place. The psychiatric conception is that the infant has only *received* until this anal phase and the first thing he is expected to *give* or contribute for the benefit of the family is evacuation at the proper time and place. Instinctively, the child expects a reward for this, his first contribution to the family comfort, which seems of extreme importance to him. Love, fondling and praise mostly will be all that is expected by the infant. The adult who reverts back to this means of attaining compensation expects much more; understanding of his problems, patience even with his illogical reasoning and so on.

If infant toilet training is done too quickly, strictly or in a punitive manner, the impression may be created that the normal elimination process is dirty, immoral and something to be ashamed of. (The result might be fear, overmodesty and apprehension in later life under certain provocating stresses.)

If economically, socially, financially, professionally or sexually frustated or unsuccessful, the neurotic patient may revert back to the infant tendency to get a reward for his gift ability, the ability to give feces.

Neuroconstipation thus could sometimes be regarded as the desire *not* to give as a defense or an anxiety to offend somebody as the infant was taught in childhood that a bowel movement could be offensive. (These latter patients often cannot urinate or defecate in presence of others.) This may be a response to anxiety, resentment or guilt and may be associated with depression, neurasthenia or over-conscientiousness (the rigid type). (It also may lead to diminution of libido.)

If *acute* physical or mental trauma occurs in later life which the patient is unable to cope with, he likewise may regress to the childhood stage—which was more pleasurable and satisfactory—even if at this time it is inappropriate or painful, resulting in elimination irregularities or other proctologic symptoms.

To quote from Dr. Karl Menninger's book, Man Against Himself, "Where responses to emotional stimuli are irrepressible, the patient falls back to the more primitive spillway of the autonomic system—'they say it with symptoms.'"

The psychologic meaning might not be recognized at a future date when the symptoms have become habitual. Occurrence of a bowel movement caused by an acute emotional experience is well known and understood by all. The continuous reaction to chronic stresses is just as real but frequently unrecognized.

I would like to say a word here about the gastrocolic reflex. If the patient can revert to his childhood to his undoing, the physician may do likewise to his patient's advantage. The gastrocolic reflex has been suppressed by training and customs of civilized social life. In the constipated patient it may often be successfully revived and retrained with resulting regular evacuations after meals.

In all these problems we should not underestimate the help of the family, especially in younger patients, and give pertinent instructions to an intelligent member as far as feasible.

The extent to which psychoneurotic symptoms could mislead the profession is illustrated in a humorous manner in a discussion by Dr. P. W. Brown, at a Mayo Clinic staff meeting in 1932, about "the organic approach of a psychoneurotic bowel." Under the title "Doctoring the Bowels" he described uncountable symptoms and innumerable, inappropriate and unsuccessful treatments and operations of a hypothetic, typical gastrointestinal neurotic

which I think every proctologist and gastroenterologist should read.

We have all believed for a long time, more so after this war, that we cannot ignore the psychosomatic aspects of proctologic symptoms and diseases and will have to enter this field more and more if we are not going to deal with local pathologic conditions only but study the patient as a whole. More extensive knowledge of this subject will enable us not only to successfully take care of many of these patients but also to select more intelligently the patients with severe forms of the disorders who definitely require psychiatric attention.

ENDOCRINE dysfunction may cause substances to enter the circulation and thus increase sensibility of sensory nerves, leading to itching in the presence of local irritation. Thus diabetes, exophthalmic goiter, and the menopause must be considered.

EVALUATION OF ANORECTAL COMPLAINTS

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IT is frequently considered that the history is of secondary importance to the physical examination and without doubt this is often true, particularly if the history is hurriedly taken and improperly evaluated. However, in every patient's story there are pearls waiting to be plucked if the physician will take the time and effort to do so.

The first two sentences in Volume 1 of Gastro-Enterology by Bockus are: "The most important part of a clinical examination of patients with complaints related to the digestive tract is a good history. Obviously, the value of the history depends upon the intelligence of the patient and the keenness and experience of the historian." The medical student is taught to take a complete and detailed history although he has little experience in evaluating its salient features. In contrast, the busy physician frequently takes so much for granted and makes the history so brief that the patient's complaints serve as a basis for his diagnosis. It appears that following internship very little emphasis is placed upon the value of the history as there is a paucity of articles on this subject and very few text or reference books contain more than the advice to take a thorough history.

Before evaluating a history a good one must be obtained. The object of the anamnesis is not simply to fill in the answers to a list of prepared questions on a printed sheet, nor to question the patient regarding his concept or judgment of the case; nor is it to inquire what he thinks is the diagnosis. The only object is to supply the physician with a set of facts, from which he draws conclusions, based upon his knowledge and experience. A good history need not necessarily be long and detailed. The patient should have the opportunity to tell his story in his own words, for he then feels he has supplied the reason for his visit. This story must be used as a base line or the point from which to seek facts regarding the patient's problem; since especially in proctologic cases, the patient is often unable to state them clearly himself. The time spent in history taking allows the physician the great advantage of sizing up his patient as well as obtaining the latter's confidence. While I am not unmindful of the necessity of personally taking a complete history of every patient, or ascertaining that this is done elsewhere, this discussion is limited to the evaluation of anorectal complaints.

Obtaining the proctologic part of a history is somewhat different from that of the general history and must be evaluated accordingly. Several reasons for this are to be considered, the first and most important of which is terminology.

Proctologic terminology is so relatively new that many patients are not familiar with our terms and, if questioned, the answers will be vague and meaningless or entirely wrong, thereby misleading the unsuspecting physician in the diagnosis. It is obvious that if patient and physician speak different languages, no history whatever is obtainable and it should be equally clear that if the physician speaks in terms which are unknown to the patient, he will not be able to give an intelligent or correct answer. Many patients hesitate to admit they are ignorant of terms used by the physician and in preference to so stating will answer incorrectly. A short time ago I heard a resident in a hospital ask a patient, "Have you had any recent inflammatory proctologic condition?" Fortunately he received the reply, "Doctor, I don't know what you mean." But how

often would a question like that bring forth the easiest way out—a simple "NO"!

We think in proctologic terminology and are very apt to assume that the patient knows the meaning of our terms which is far from the truth. It is not too unusual for patients to deny having hemorrhoids, but to admit having "piles"; in fact, a great many patients' only conception of anorectal disease is "itching and bleeding piles." Also, consider the relatively old and familiar word "diarrhea"; how many patients know exactly what is meant by this term? It is too varied in its interpretation to be used directly to a patient, for what is "diarrhea" to one, has an altogether different meaning to another. Rather, receive an immediate and accurate reply to the query as to whether the patient ever has had four to twelve soft, watery stools daily. This correct answer may then be placed on the history sheet after the word "diarrhea" if the printed form is used. Therefore, since the patient is unable to interpret scientific terms of the proctologist, the latter must question the patient in his own language and in turn evaluate this in proctologic terminology. If the keenly alert and tactful physician suspects this to be the case, he will grasp the opportunity to assist with well chosen words, allowing the patient to select those which best suit his symptoms. This initiative will be productive, not only of a good and accurate history, but also of a grateful patient; otherwise he may leave the office with the impression that the physician is at fault for not diagnosing his case.

The patient is usually more reluctant to state anorectal complaints to the general practitioner than to the proctologist because, by the time he reaches the office of the latter, he is mentally prepared either by the first physician or by the severity of his symptoms "to go through the worst." Even so, many patients find it difficult to state frankly their complaints to the proctologist. The most usual reason for this is embarrassment since the patient regards his anorectal area as his private property and its examination, either by the patient himself or by the physician, is uncommon. This feeling is enhanced by the fact that the rectum and anus act as a passage way for body excretions, and, in the presence of pathologic disease its cleanliness is difficult and uncertain. Patients frequently show this attitude by their remarks that they dislike to ask a physician to make what they consider an unpleasant examination, forgetting that the physician's interest is one of ascertaining facts regarding his patient.

Not only is the patient reluctant to make complaints of anorectal distress and submit to a proctologic examination because of modesty, but frequently fear of pain is a contributing factor. This may arise from his own personal experience with a painful anal ulcer, a cryptitis or external thrombotic hemorrhoids, or may be present because of the many and usual stories regarding painful anorectal examination, treatments or surgery. Whether or not justification for this view exists, it is a definite factor in many instances for withholding information regarding anorectal symptoms. To overcome this attitude requires tact, reassurance and patience on the physician's part and time spent in this way pays big dividends during the examination.

To be of value, a properly taken history must contain facts which are usually established only by careful questioning. If one accepts the history as told by the patient, much will be omitted because of the limited vocabulary, the intentional or unintentional oversight of some symptoms or the patient's poor recollection of forgotten ones. These omissions may very well contain the crux of the story and will be obtained only by careful and persistent questioning by the physician; in truth, this may be compared to an oral examination or even a cross-examination which implies a keenness of insight into possibilities and a persistency of inquiry into details to obtain all the facts present. To have a productive line of inquiry the

physician must be trained to think in terms of anatomy, physiology and pathology. In this manner his reasoning will quickly correlate the patient's complaints with known possibilities and his inquiries will be guided along logical lines and in the proper direction. Thus, there rapidly will be obtained a maximum of pertinent facts and a minimum of irrelevant material. Accepting at face value statements made by patients becomes a matter of accepting their judgment. The latter depends upon intelligence and experience and most patients have relatively little proctologic knowledge or experience and therefore cannot be expected to form correct opinions.

A few months ago a patient told me she had consulted a physician with the complaint that she had "bleeding and painful hemorrhoids." The busy physician immediately told the nurse to put the patient on the table and he would treat her hemorrhoids which he proceeded to do without further history or examination. The patient really had an anal ulcer but she made the only diagnosis she knew and, unfortunately, the physician accepted her faulty judgment as a fact.

In another instance, following the removal of a couple of skin tags, a patient called by phone at night, stated that she was having a severe hemorrhage and demanded admittance to a hospital. Upon arrival there only a small amount of bleeding was present although the patient persisted in the belief that she had had a severe hemorrhage. The color of the skin, pulse, blood count and blood pressure in conjunction with the local examination belied even any moderate hemorrhage. This then was only the patient's judgment.

These two illustrations could be multiplied by any of us and would serve only to center our attention on the truth of the statement that a patient's judgment of anorectal pathologic symptoms is unreliable. One is impressed by the frequency with which patients arrive at false conclusions because of lack of knowledge and experience or because of preconceived notions. The physician must be on his guard against accepting these without due proof.

Referred symptoms are most difficult for the patient to recognize and interpret as well as for the physician to discover and evaluate. The patient usually is unaware of the possibilities that rectal or colonic disease may cause symptoms in other parts of the body and cannot be expected to appreciate the value of them in relation to their proctologic complaints. Consequently, they are often overlooked by the patient when he tells his story. But in searching for all the facts which can be elicited, the alert physician will be cognizant of these referred symptoms knowing that the rectum and colon are innervated by the sympathetic nervous system and consequently prone to present referred symptoms. The correct interpretation of these is essential and of inestimable value in directing the examination along productive lines.

In conclusion, it may be stressed that proctologic complaints can be evaluated properly only after obtaining all the facts present by the purposeful questioning of the patient; that this, together with physical examination and indicated laboratory work will lead to a correct diagnosis, proper treatment and a satisfied patient.

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DISCUSSION

Knox Brittain (Rochester, N. Y.): I agree with Dr. Brown's value of the history in proctologic cases. On the other hand, it does not displace or replace complete, adequate and thorough physical examination. Anyone who tries to make a diagnosis with either the history

or the physical examination alone fails in making a correct diagnosis. Certainly an opportunity is given to the physician in taking the history to study the patient from a psychological standpoint and the patient is given an opportunity to tell his story.

Dr. Brown emphasizes the confusion of the layman on hearing proctologic terms. Certainly there is a good deal of confusion among us in the use of terminology. I think it cannot be too greatly emphasized in paying attention to the patient's complaint. For instance, a patient comes in and says he has pain and a routine, possibly a little bit careless examination fails to discover cryptitis, whereas a bidigital examination does reveal actual pathologic symptoms. In other words, there are big stores like Sears, Roebuck who tell us the customer is always right. Possibly it is a good theory for us to follow. Very often, for instance, the patient will tell us they have had bleeding and we find that they have hemorrhoids which continue to bleed in spite of either operation or treatment. If we persist in examination, repeatedly examining that patient, we find that they do have a small neoplasm. I believe as Dr. Brown emphasizes, that any complaint worthy of bringing the patient for examination is also worthy of a complete and adequate examination.

Charles C. Massey (Charlotte, N. C.): Quite naturally, evaluation of proctologic complaints is most important, for here we initiate a procedure which usually leads to the pathologic condition we are to find and to the medical remedial measures we are to employ. In the proper evaluation of a patient's symptoms, we must consistently apply ourselves as seriously as we do to our operating room technic; for, after all, a good result means satisfaction both for the patient and physician.

I enjoyed Dr. Brown's paper and find little more to add to the discussion.

PYRIBENZAMINE

ITS RÔLE IN THE TREATMENT OF PRURITUS ANI

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New York, New York

COMPARATIVELY few individuals suffering from diarrhea, prolapsing hemorrhoids, chronic discharging fistula, incontinent sphincter, etc., complain of pruritus ani despite the insult to the exposed integument by fecal material and malodorous infected discharge. The perianal region of over 1,000 young men examined for the draft showed contamination by varying amounts of excrement and yet very few complained of itching.

Is it not logical to assume that in pruritus ani there must be two contributing factors? (1) Sensitization of the skin; (2) an irritant (allergen or antigen). If this premise is true, then the pruritus must, in the main, be an allergic phenomenon.

"Recent investigations[1] have shown that all individuals are potentially capable of developing allergy. The capacity to become allergic depends on two fundamental factors: (1) The predisposing or auxillary condition[1] which paves the way for the allergy by making the organism prone to allergization. (2) The exciting allergens, in the case of food hypersensitiveness, the nutritional allergens which actually elicit the allergic reaction.

"Such predisposing factors are hereditary, the functions of the endocrine glands and of the autonomic nervous system, infection, intoxications, gastro-intestinal disturbances, hepatic disease, infestations, metereologic and climatic influence, social and environmental conditions, psychic influences, vitamin deficiencies."

A comprehensive history, intimate study of the involved integument, supplemented by skin tests if necessary, will determine to what group the exciting allergen belongs. The following questionnaire[2] is used in our practice:

I. Does the patient know of anyone in his family with: (a) asthma? (b) hay fever or vasomotor rhinitis (seasonal occurrences)? (c) atopic dermatitis including infantile eczema? (d) seborrheic conditions (baldness, acne, seborrheic dermatitis)? (e) hives—angioneurotic edema (giant hives)? (f) other skin eruptions, including those due to drugs? (g) other untoward effects from drugs? (h) other familial diseases?

II. Has the patient had: (a) asthma (not cardiac)? (b) hay fever? (c) stopic dermatitis (including "infantile eczema")? (d) fungous infections? or eczematous "ids"? (e) poison-ivy dermatitis? or other contact-type dermatitis? (f) seborrhea: 1. dandruff? 2. acne? 3. falling hair?—baldness? (g) hives? (h) other skin eruptions? (i) other diseases?

III. (a) When did the eruption begin? (b) Was there a previous similar attack? (c) Where did the eruption begin and how did it spread? (d) Is the eruption constant or periodic of sudden or gradual onset? 1. seasonal influences? 2. Other remarks on course of dermatosis. (e) Is there anything which has been recognized as making the skin condition worse? 1. foods? 2. clothing? bedding? articles of furniture? dusts? sprays? particular places of work, of residences, etc? 3. menses? 4. illnesses and remedies? 5. miscellaneous? (f) What treatment has been used—internal and external medicaments? (g) Medication in general (proprietary or prescribed): 1. for headaches? 2. for constipation? 3. for "acid in systems" (blood purifiers," tonics)? 4. for sleeplessness? 5. for "nervousness"? 6. for coughs? 7. for dysmenorrhea? 8. for "sinus trouble"? 9. Bromo Seltzer? Bromo Quinine Laxative? Ex-Lax? phenolphthalein in any other forms? other "patent"

remedies? home remedies? medication on recommendations of relatives or friends, of druggists, of previous physicians? (10) iodized salt? (11) other medication—internal or external, proprietary or prescribed? (h) Cosmetics 1. used by the patient? 2. used by others in the patient's environment? 3. applied at beauty parlors or elsewhere? (i) Occupational and avocational contacts: 1. At work (a) principal substances handled? (b) incidental substances handled (cleansers, washing materials, "protective" materials, etc.)? (c) physical condition of the contacts? (heat, moisture, friction) 2. At home (a) cleansers, insecticides (professional exterminators), (b) pets, plants and substances used in their care, (c) clothing, furniture, drapes, (new-cleaned—dyed)? (d) hobbies—photography, stamps, carpentry, gardening, golf, cards, other games? (e) miscellaneous? newsprint, rotogravure, jewlry, nickel-plated articles, rubber articles, etc., etc.,

Summarizing this history form, the exciting allergen will be found to fall into one of the following groups:

1. Allergy to food: Shell fish, fish (including caviar, etc.), strawberries, cheese, nuts, eggs, wheat, milk, pork, chocolate, cocacola and alcohol in all its forms, etc.
2. Allergy to fungi and bacteria: Trichophyton, epidermophyton, or monilia albicans, any bacteria.
3. Contact dermatitis: (Lesion primarily epidermal; spongiosis, vesicle, papule, etc.) dyes, silk, wool, perfume, toilet water, talcum powder, cleaning fluid, drugs and ointments applied to the skin, leakage of mineral oil with fecal material, feces, clorox used in bleaching clothes. Sensitivity to fecal material is probably due to skatol, indol and complex phenols. The medicinal agents applied in extended postoperative care will often cause a contact dermatitis. Benzocaine is a frequent offender.
4. Allergy to drugs: Any drug taken internally may cause symptoms. Such drugs are ipecac, quinine, barbiturates, opiates, iodides, including iodized salt, aspirin, bromides, antipyrine, sulfa compounds, phenolphthalein, etc.
5. Allergy to atopans—atopic dermatitis: Specific circulating antibodies can be demonstrated by passive transfer experiment of Prausnitz and Kuestner. A complex of symptoms and phenomena which can be explained primarily by increased vascular permeability. A family history which is positive for asthma, hay fever, atopic dermatitis and perhaps certain other diseases such as migraine, hives and gastrointestinal manifestations.

A personal history which often reveals, in addition to the presenting atopic disease, certain other atopic manifestations—mentioned above. An eosinophilia in the blood smears and/or in the fluids and tissues of the affected parts. A specific substance (called atopen) which reaches the sensitized cells in sufficient quantity. (The atopens may be inhalants, such as: pollens, spores of fungi, emanations from animals, insects, etc., dusts and powders, orris root, rice; ipecac, caroid and lycopodium in prescriptions filled by druggists, wheat flour, etc., either by inhalation, swallowing or by external exposure and transepidermal penetration of the outer skin.) Cosmetics, insecticides, etc. Injected substances—serums-vaccines, etc. Microorganisms including bacteria and fungi. Drugs (aspirin, etc.). Foreign proteins. (All these can act by any route provided they reach the specifically sensitized tissue in sufficient quantity per unit of time.)

The allergic individual has sensitizing antibodies circulating in his blood. These are also attached to the body cells of a certain organ known as the shock organ. A foreign substance (irritant, allergen, antigen) is introduced to the skin or in the body which interacts or probably combines with the antibody liberating histamine proper or a histamine-like substance caus-

ing the allergic reaction by breaking off a CO or carboxyl radical from histidine, one of the body amino acids.

There is hardly a tissue that will not respond in some way to histamine.[3] This drug is a powerful dilator of capillaries. As a result there is a loss of plasma protein and fluid through the capillary wall into the extracellular spaces. This phenomenon is most important in pruritus ani. All histopathologic studies of pruritic skin emphasize marked dilatation of the blood vessels.

"PYRIBENZAMINE (N'-Pyridyl-N'-Benzyl-N-Diethyl-Ethylenediamine HCL) was discovered by Mayer[4] and his associates. It is a white crystalline material which is stable, non-hygroscopic and readily soluble in water. It can be safely administered in dosages up to 150 mg. four times a day preferably after each meal and at 10 P.M.

"PYRIBENZAMINE is well absorbed[5] from the gastro-intestinal tract, peritoneal cavity, subcutaneous and intramuscular depots. Its oral administration is so satisfactory, that the risk of unwanted side reactions from parenteral administration is unwarrantable. Nothing is as yet known of its destruction, deposition or secretion."

Its use over a considerable period of time has as yet not evidenced any effect on the blood, urine, liver or other vital organs.

As to the action of pyribenzamine, I should like to quote from a letter received from the Research Department of the Ciba Pharmaceutical Products:

"Frankly, we do not know exactly how pyribenzamine acts as an anti-histaminic agent, but it is safe to state that pyribenzamine and histamine enter into a competitive relationship in that they compete for the site of action normally involving histamine whether it be in a muscle, glandular or other type of cell. We speak of this phenomenon as that of competitive inhibition and you are correct in your statement when you say that pyribenzamine acts better when employed prophylactically than correctively; in other words, an ounce of prevention is worth a pound of cure seemingly.

"Pellerat[6] has demonstrated that after the administration of antihistaminic agents to experimental animals not only is anaphylaxis prevented but histamine is liberated in abundance. This indicates that histamine does not seem to be chemically neutralized or physico-chemically fixed with the anti-histaminic agent but rather that it is prevented from eliciting its normal physiologic reactions on the part of the system due to the fact that it apparently is insulated against by pyribenzamine or another antihistaminic agent at the site of its normal action whether it be at or within the cells involved. This insulation, probably similar to that afforded the electrician against electrical currents by the use of the rubber glove, might be purely mechanical in the sense that a protective physical relationship is established at the surface of the cell, but in the light of modern biophysics and biochemistry, one would anticipate that certain enzymes might be involved in this relationship. What these enzymes are in problematical and only time will permit elucidation of this phenomenon."

Dr. Rudolph Baer, of New York, gave a quantity of this drug to my wife in 50 mg. tablet form to alleviate a distressing, itching, allergic foot condition. The tormenting itching and swelling was promptly relieved.

If this new agent were to control the itching of the majority of patients with pruritus ani, the topography of the skin would improve because the trauma of scratching would be removed. Sleepless nights would end, the patient's health and morale be bolstered and skin therapy would provoke a greater response. It would above all demonstrate that the pruritic skin lesion in the perianal region and its environs is the result of an histaminic action otherwise why could it be controlled by an anti-histaminic drug? Since most allergists agree that reaction in the receptor cells of the shock organ has to do with a histamine effect, such a response to specific therapy would prove our premise that pruritus ani is in the main an allergic manifestation.

This research with pyribenzamine began in July, 1945. The supply of the drug was limited at that time so that our experiment began in earnest in November, 1945.

A careful history was taken from each patient using Sulzberger's questionnaire. The patient was instructed to write down each day foods consumed, drugs taken and note particularly if anything partaken internally or used externally influenced the itching. If the incriminating allergen could be determined, it was eliminated.

Patients with pruritus ani were advised to take 50 to 150 mg. of pyribenzamine after each meal and at 10 P.M. They were warned against any other medication for at least forty-eight hours. Placebo tablets provoked no relief when prescribed. They were not told what the action of this new drug would be so that the psychic effect could be discounted.

If drowsiness occurred, the dosage was reduced. Drinking black coffee would usually overcome this symptom. If any untoward syndrome developed, the drug was discontinued.

Dosage could be reduced to one tablet a day. If the individual complained of nocturnal pruritus, an appropriate quantity was given after the evening meal and before retiring. Gradually, one tablet before retiring was frequently sufficient to insure an uninterrupted night's sleep.

Because Gerhard Katz[7] and other observers were able to demonstrate histamine in allergic skin lesions an attempt was made to evaluate the effect of a 2 per cent pyribenzamine ointment* in a water washable base, applied to the involved area.

When first applied, the ointment causes a smarting sensation lasting about one minute. If it is effective, the itching should be controlled within ten minutes. In this survey it was revealed that only those patients that responded to pyribenzamine taken orally obtained relief when the ointment was applied to the skin. After a time the itching could be controlled by local therapy alone.

* Supplied by The Ciba Company.

Our best results were obtained using a combination of oral and local medication. Adequate doses given by mouth yielded better results than when only the unguent was used. Patients with an acute, exudative eczema of the perianal region were not treated with this ointment.

If the incriminating allergen had not been ascertained, further studies were made but in the interim the patient was given the following instructions:

(1) Omit from the diet anything sour, bitter, salty, condiments, flavors, coffee and all forms of alcohol. Mineral oil or oil base medications must be avoided.

(2) After defecation wipe perianal region with wet absorbent cotton, sprinkle acidolate on cotton and wash the affected area (to remove sweat, fecal material and ointments). Wash off acidolate with another portion of wet cotton and then dry with absorbent cotton. The area is then covered with either lotion, unguent or powder, depending upon the skin topography. Always keep the area covered with a thin layer of fine, absorbent cotton. Some may argue that this cleansing is responsible for the relief and cure of the pruritus. In my series this was true in only six patients.

(3) Do not take tub baths, only showers.

(4) If interdigital epidermophytosis is present, soak feet in hot water for ten minutes to remove sweat and dye from stockings. Dry feet thoroughly. Apply liquid sopronol on an applicator between the toes and to other affected portions of the feet. This should be done at night. In the morning, dust powdered sopronol between the toes and over the affected areas. Wear white cotton stockings. A separate towel should be used to dry the feet so as to avoid transmitting infection to the perianal region or groin.

(5) In patients with acute exudative eczema or when the itching is tormenting in character, ice in a throat ice-bag applied to the perianal region will usually give relief in fifteen minutes. In those cases in which there is weeping, a 1 to 20 liq. burowii compress is applied to the skin and the ice-bag

placed over it. This freezing agent must be pressed closely to the compress to avoid a column of air between ice and skin. A domeboro tablet dissolved in a pint of water will make a fresh 1:20 solution. Application of ice numbs the nerve ends and constricts the capillaries and arterioles. When the acute skin phase becomes subacute or chronic, appropriate lotions, unguents or powders are applied.

(6) In patients with contact dermatitis from feces, cover the perianal skin with zinc oxide ointment before evacuation. After the bowel is emptied and the perianal region washed with acidolate cover the integument with 1 per cent methol in equal parts of zinc oxide ointment and desitin ointment.

(7) If skin is infected or undermined, supply adequate drainage.

(8.) All clothing or bed linen coming in contact with the perianal skin should de washed in lux. Clorox or other bleaching agents used by commercial launderies frequently cause contact dermatitis.

Young women suffering from profuse vaginal discharge were routinely examined for trichomonas vaginalis. If a history of pinworms is elicited, appropriate therapy should be instituted. Urine is examined for sugar. Metabolic diseases, vitamin deficiency, etc., are adequately treated.

STATISTICAL DATA

Ninety patients were treated with pyribenzamine. Fifty-seven had satisfactory results (85 to 100 per cent had relief from itching); twenty-six cases were due to food allergy; ten cases were due to fungous allergy; twelve cases were due to contact dermatitis; three cases were due to atopic dermatitis and six cases were due to drug allergy. Eleven patients reacting favorably to the oral administration of the drug were controlled by the local application of a 2 per cent pyribenzamine ointment in a water washable base. This ointment was only available for the last few months of this survey.

Thirty-three patients had unsatisfactory results. Sixteen did not return after their first visit, the investigation was incomplete and the response to therapy was uncertain; there were four patients in whom the drug had to be discontinued; two patients developed a generalized maculopapular eruption; one patient complained of cardiac palpitation and extreme nervousness; one patient developed very profound drowsiness; six patients with contact dermatitis were not controlled; in the four patients with senile pruritus three responded to estrogenic and androgenic therapy; one patient was diabetic and two patients had drug allergy.

There were two patients with psoriasis principally involving the posterior aspect of the perianal region and extending toward the coccygeal area. The only other phase of this disease was found under the folds of the mammae. These patients were relieved by using a ½ to 5 per cent ammoniated mercury ointment and finally healed with a 3 per cent vioform unguent in a water washable base.

Arbesman[8] and his associates were the first to conduct therapeutic trials with pyribenzamine. Feinberg[9] reports excellent results in controlling the itching of pruritis ani and vulvae.

CONCLUSIONS

1. Anorectal pathologic conditions play an insignificant rôle in the etiology of pruritus ani.

2. Pyribenzamine will not cure this syndrome.

3. Pyribenzamine will control itching in the majority of patients. By reducing sleeplessness the morale and general health of the afflicted person improves. Since the trauma of scratching is removed, the pathologic condition of the skin improves and adequate therapy can be instituted (lotions, ointments, powders, etc.).

4. The percentage of patients with pruritus ani due to fungous infection is not as high as anticipated. Because there is an interdigital epidermophytosis in the same individual, it does not prove that the skin

lesion in the perianal region is necessarily of the same origin. It is the exception rather than the rule for the patient with athlete's foot to suffer from pruritus ani.

5. Careful investigation will clarify the etiology of many patients heretofore classified as idiopathic.

6. If the incriminating allergen has not been ascertained, it is due either to the fact that the investigation was incomplete or because our methods of determination are thus far inadequate.

7. Because a considerable percentage of patients do not respond to pyribenzamine it does not rule out their allergic origin. The logical explanation must be that the pyribenzamine is unable to insulate the receptor cells in the shock organ against the action of histamine. This fact is further borne out by the more satisfactory results obtained in animal experiments when pyribenzamine is administered prior to exposure to the offending allergen.

8. The degree of success in the alleviation and cure of pruritus ani will depend on the thoroughness of the method of investigation plus a fundamental knowledge of allergy, dermatology and proctology.

9. Pyribenzamine merits a definite and important rôle in the treatment of pruritus ani.

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RATIONAL OF THERAPY IN PRURITUS ANI

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WE are all hoping that some one of us will rise and deal pruritus ani one fell blow that will knock it out of the ring but to our sorrow the time is not yet at hand. There is such a maize of interlacing and overlapping of factors that even cause and effect become reversible.

Pruritus ani as it confronts the proctologist is not a mere symptom but constitutes a definite syndrome[1–4] and along with the recurrent attacks of itching in the anoperineal region, we are involved with the associated symptoms of anospasm, insomnia, nervous depression and sluggish peripheral circulation; also excessive perspiration in intertrigenosis areas, a moderate leukopenia and a subclinical nutritional deficiency state and, of course, the gamut of skin involvement.

Whether or not we accept pruritus ani as a syndrome we are all, nevertheless, aware of the coexistence and importance of the associated findings. Our rational of therapy, therefore, must include the consideration of all these factors.

The nervous manifestations are so characteristic that we can easily describe a type that would represent a cross section of these patients. They look, as a rule, well nourished but pale and pasty with a grayish cast to their skin. Even when thin, they are sluggish and slow moving with lackluster eyes. They are generally not communicative but give an impression of dejection at times amounting to a state of despondency with suicidal tendencies. These nervous manifestations represent a vagosympathetic imbalance of the vagotonic type.

In order to appreciate the psychosomatic factors of this syndrome we must review the nervous mechanism involved. It is true that the itching sensation travels by way of the sensory or pain fibers, however, the threshold of irritability for receiving the sensation involves the vegetative (autonomic) nervous system so that when the vagosympathetic equilibrium is upset, as in vagotonia, a severe pruritus may be elicited by ordinary innocuous stimuli. Furthermore, since the psychic factor is the true receptor of the sensation, its state of equilibrium will in turn determine the degree of response it will give to the sensation of itching.[5]

Any therapeutic measure, therefore, that tends to calm the psychic state or reduce the nervous irritability[6] or tissue sensitivity enters as a valuable factor is the treatment of pruritus, whether it is bromides, barbiturates, phenobarbital or calcium or the more recent anti-hystamine preparations like benadryl and pyribenzamine.

It is further established that some of these states showing autonomic imbalance have their basis in vitamin B deficiency.[7,8] I have long recognized the importance of these factors, so much so that when I find the pruritus associated with a vitamin B deficiency I consider the problem half solved.

The basic pathologic condition consists of three factors: skin irritation, anospasm and lymph stagnation or lymph edema. These three factors are so completely interlaced that you cannot separate any one from the others. Together they constitute both the cause and effect of neurobegetative imbalance and form the groundwork of this syndrome.

RÔLE OF LYMPH STASIS

Lymph stasis brought on by the slowing of the peripheral circulation plays an important rôle in the chronicity of pruritus. It allows the further accumulation of the

irritating substance whether it is excessive or abnormal products of metabolism or actual toxins.

Hanes[9] began his local injection procedures when he recognized that there was some disturbance in the cellular metabolism in the local tissue. He described the almost immediate correction of advanced skin changes following his injections in the subcutaneous tissues. Those of us who have used the Hanes procedure have witnessed the disappearance of even remote coexisting skin lesions following the injection of hydrochloric acid solution in the perianal region.

Our injection methods, even those that primarily anesthetize the sensory nerve endings, all produce a phagocytic response and basically have the same physiologic effect of decongesting the peripheral circulation. The phagocytic activity of the histeocytes produced by the reaction to these injections are known to remove toxins and protein particles as well as bacteria so that they may account for the major part of the readjustment of peripheral lymph circulation.

Another very probable factor is the interference with adequate oxygenation of the tissue cells by the slowing of the circulation. This may be the reason for the good results obtained by direct injection of oxygen as described by Dr. Guess.[10]

We see, therefore, that there is a common denominator in the rational of all our injection treatments.

RECTAL PATHOLOGY

We all agree that the more common rectal conditions are seldom directly responsible for the itching. However, the most constant rectal finding in all these patients is anospasm. Any indication of a rectal pathologic condition that will produce or intensify rectal spasm should be corrected for if it is allowed to remain this condition alone can eventually bring about neurovegetative exhaustion.[11]

It is always best to postpone surgery until we can determine the underlying factors that are interfering with the normal physiologic balance. Proper timing of drastic procedures is also of value for the morale of the patient. It is important in case of recurrence to prove to the patient that the itching can be relieved by simple measures and that our clinical resources are not easily exhausted. However, my only failures are those when I underestimated the despair of the patient and waited too long before instituting drastic measures for his relief.

In frustrated individuals who are facing insurmountable problems and in patients who have received x-ray therapy, it is best to give immediate symptomatic relief and proceed later with general measures to correct basic underlying factors and with instituting measures to prevent recurrence.

However, I see no justification for radical surgical procedures *per se* and injections involving mass tissue destruction. I do not even use the undercutting type of operations because I find that alcohol injections by the multiple puncture method of Stone[12] and Wilson[13] will accomplish just as much. It is rarely necessary to repeat the injection more than twice.

Whenever there is scar tissue, redundant folds, pectinosis, marginal varicosities or ulcer scars with thickened margins, I find it advisable to perform surgery and use alcohol stippling to prevent recurrence of pruritus immediately following operation.

RÔLE OF FUNGUS INVASION

One of the features of this syndrome is the excessive perspiration of the apocrine sweat glands, producing an alkaline secretion rich in protein and carbohydrates. The location of these apocrine glands are also and probably therefore the site of predilection for cultivation of fungi. Since they are saprophites, they are also furthered by the basic interference of the cellular metabolism by the lymph edema described above producing partially devitalized cells more suitable for their saprophitic comsunption.

Once they arrive the fungi entrench

themselves by means of their alkaline secretion and perpetuate and intensify the very same cultural conditions that made their arrival possible.

It is very difficult to isolate the fungus organism from the perianal skin once treatment has been instituted.[14] An important factor to take into consideration is the seasonal tendency of recurrence: Not only is there a much greater incidence of cases during definite periods but the organism can be detected much more readily during that time. The beginning signs of fungus pruritus is the linear abrasion or scaling in the intergluteal fold. I call this the "tell-tale line." Another tell-tale sign is the thickening or lymph edema of the anterior median raphé in the male.

In advanced cases as we all know the skin undergoes a great variety of changes. We must remember that conflicting medications can give rise to an "hysterical skin" so that on first inspection it will not fit into any category. At this stage it is best to use the so-called bland and soothing medications such as boric acid ointment. At times the irritation is so extreme that the skin will exhibit a non-specific sensitivity reaction to almost all topical medications so that only bland oils will be tolerated. This is a critical point in the progress of the patient with pruritis. I call this the "crisis." It is at this stage that the patient becomes frantic and changes doctors or the doctor becomes frantic and resorts to irrational procedures.

We must remember that all fungicidal ointments owe their effectiveness to their acid nature. However, fungicidal ointments that have a heavy petrolatum base will only plug the hair follicles and sweat glands of the perianal region and will not reach the fungi. Such an ointment, although most effective in other parts, will only cause further irritation of the perianal skin. In these instances it is best to use penetrating dyes.

In conclusion I want to emphasize that we must accept the rôle of the clinician in addition to treating the rectal pathologic condition if we want to track down and irradicate pruritus ani tendencies. I credit my good results to the fact that I accept pruritus ani on its own terms and give all features of the syndrome equal respect.

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DISCUSSION OF PAPERS OF DR. FRANK M. FRANKFELDT AND DR. RACHELLE SELETZ

Robert V. Terrell (Richmond, Va.): While in general agreement with the views expressed by Drs. Frankfeldt and Seletz I wish to re-emphasize my own belief that the cause of pruritus ani is in most cases a mycotic infection of the perianal skin. I readily concede that allergy plays a part in many patients just as a neurosis undoubtedly is a factor in a few.

Best results will be obtained by modifying the treatment to suit the condition of the perianal skin and the character of the patient. Should an allergy or a neurosis be present, efforts are made to correct these while treating the skin. After a painstaking proctologic examination considerable time is spent in explaining to the patient in language he can understand just what is wrong with him and how we shall attempt to correct the condition. The importance of his cooperation is stressed, explaining that he should use only drugs prescribed to prevent the possibility of a drug dermatitis. He is also told that treatment must not be discontinued as soon as the itching ceases but kept up until the skin returns to normal.

The patient understands well enough that scratching aggravates his condition and if it is thought that local applications will not adequately curb the desire to scratch, a few cc. of 0.25 per cent diothane hydrochloride are injected beneath the involved skin. Relief from itching after this procedure is usually prompt and lasts several days. Every effort is made to improve the condition of the skin while this relief lasts.

Many patients when first seen have an acute inflammation or show a drug dermatitis. It is important in this type to eliminate any irritant and use soothing measures such as boric acid ointment or even hot boric acid compresses. Pyribenzamine is particularly useful in cases of this kind. Careful cleanliness is important in all cases but soap and water are best avoided in exudative conditions.

In the more chronic types, 2.5 per cent undecylenic acid or a diluted Whitfield's ointment may be applied three times daily. A few fibers of dry cotton interposed between inflamed skin surfaces will reduce friction and irritation and absorb serious discharges.

When pruritus ani is complicated by fissure, fistula or hemorrhoids these conditions are corrected by surgery. The patient is made to understand that we are not operating primarily for the pruritus even though we do expect it to improve. Treatment for pruritus is continued after the operative wounds have healed. We have never thought it necessary to undercut the perianal skin.

Harry C. Guess (Buffalo, N. Y.): The person discussing a paper, or in this case papers, has two alternatives. He can agree and comment or disagree and augment.

Pruritus ani, our perennial visitor, is a large and one of the most interesting subjects in proctology. For many years each year something new has been added to our already large armamentarium for the treatment of pruritis ani and often each one who offers, and some few followers, are lulled into a complacent state that the last word for pruritis ani has been spoken. Like religion striving for the heavens, some by water, some by air and some by other means, we too continue to strive for the best "in the end."

Dr. Seletz voices the hope that quote, "someone of us will rise and deal the final blow to pruritus ani," unquote. We concur in that hope. There is no doubt that we agree on the associated clinical findings in most cases and our therapy must include the consideration of all factors. That there is lymph stasis as well as arterial and venous congestion is factual.

Her timely suggestion to limit radical surgery is well given and accepted. There is probably no one condition that has so many remedies offered as does pruritis ani. Her description, as the result of the use of many of these remedies, of a "hysterical skin" is well presented but to me some are not only hysterical, they are crazy. Her conclusion that the clinician "should track down and eradicate pruritus and its tendencies" is also excellent.

As for Dr. Frankfeldt's paper my knowledge of allergy is limited and I make no claim to understand the various phases of that condition. It is said that it is better to remain silent and be classified as dumb than to speak and remove all doubts. His is a contribution for the use of one antihistaminic drug with which he has good results with the help from chemo and low temperature therapy. He states that there are two contributing factors, namely, (1) sensitization of the skin and (2) an allergen as the principle cause of pruritus. He states that if this premise is true then pruritus must be, in the main, an allergic phenomenon. That is the sixty-four dollar question as yet unanswered. I cannot agree with this statement except in some cases. My concept regarding the pathologic conditions causing pruritus ani and our results from various medical and surgical treatments reported previously over a period of years is, I believe in the *main*, the cause of pruritus ani.

That end oxins, allergens, histamine and the end products of the metabolism, whether it be Carbon No. 1 or Carbon No. 13, do cause irritation of the tactal corpuscles in the nerve endings is true and an anoxic state results. These end products of metabolism no doubt remain *in situ* because of the slow lymph arterial and venous drainage.

Regarding the statistics which Dr. Frankfeldt quotes, I have had some experience with the antihistamine product in question. Much work was done by men from my home town on hay fever, asthma and urticaria but very little with pruritus ani, and the results which stated that twenty-seven or twenty-eight patients with pruritus ani were markedly improved by this antihistamine drug were reported by a specialist from some other city. Request for

information regarding these patients was not forthcoming because the contributor had not released his paper to the press. In the latest reports of this drug, pruritus ani is not reported. However, Dr. Frankfeldt in making his conclusions does not claim the utmost for the drug but states (1) "That pyribenzamine will not cure pruritus ani;" (2) "will control itching in the majority of cases" and (3) "that it has a definite and important rôle in the treatment of pruritus ani." With these statements we must agree except for the adjectives describing the rôle and the treatment.

To conclude, both contributors have given food for thought and are to be commended for their investigations. We should keep in mind at all times (1) That we must not accept each new drug without sufficient evidence that there is value to the drug mentioned; (2) that we, as proctologists, should have an open mind in the diagnosis and treatment of this condition and (3) that some day we, as an organization, will correlate with the physiologists, pathologists, psychiatrists and biochemists and bring to a realization the ultimate for which we are all striving—that "final blow to pruritus ani."

Howard K. Belnap (Ogden, Utah): In discussing the papers of Dr. Seletz and Dr. Frankfeldt, Dr. Terrell and Dr. Guess have covered the subject as I would cover it very accurately. I am of the opinion that had Dr. Frankfeldt had a little more time in presenting his paper you would have heard a regimen of therapy that was very meticulous; it was bland; it was relatively non-irritating to the skin but I am sure that with or without the use of pyribenzamine, he would have obtained as good results as he is doing with pyribenzamine.

I want to reiterate that he does not attribute pyribenzamine as a cure, only as a relief of the itching; yet any drug that will relieve 77 per cent of patients with itching within twenty-four hours is worthy of trial. Pyribenzamine was first introduced to me in January. Like all of the other remedies that we find are at present on the market and as articles appear in the literature, we grasp at them as though we were a drowning man grabbing at a straw in a stream for we have as yet not found a specific therapy for the disease, pruritus ani. Since January, I have used pyribenzamine as directed in the literature and as outlined by Dr. Frankfeldt. My results have not been as striking as Dr. Frankfeldt's. I have obtained relief of itching in about 13 per cent of my patients. Maybe I did not use as much of the drug as was necessary to disrupt those inhibitor factors that Dr. Frankfeldt talks about but I used the prescribed milligrams four times a day.

Again, let me repeat I think his good results have been from his other therapy.

In Dr. Seletz's paper, the discussion that it is a vagotonic, vagosympathetic balance is really something to consider. She has brought forth a point that we have, as proctologists, been tending toward in the last two or three years. As you look over the review of papers presented, we are connecting up the sympathetic and the parasympathetic systems with the symptoms and conditions we find about the rectum. Her discussion is a step in that same direction.

She, again, outlines a regimen of therapy that is good. It follows along the same outlines as we generally apply. Whether she looks at all the other symptoms and treats each one of them with an individual pill or with some individual type of psychotherapy, that is a problem and her method of therapy and it should be good.

I agree with Dr. Terrell and am in accord with him to the extent that I believe pruritus ani has its basis in a micotic organism. In my patients I found either a trichophyton or one of the epidermal phytons in scrapings of the perianal skin. I think that sometime in the future when we are able to obtain a fungicide that will penetrate through the entire corneal layers of the skin without producing a deleterious effect, we will definitely come to some conclusion in the treatment of pruritus ani.

Malcolm R. Hill (Los Angeles, Calif.): My short discussion will be brought up in the light of a few remarks alluding to Dr. Terrell's premise that mitotic origin of pruritus ani still stands in the foreground. My groundwork on pruritus ani was obtained from Dr. Emmett Terrell years ago. It was on his remarks to me as an individual and placing that premise in the foreground that a study and report was made to this organization at the Richmond, Va. meeting several years ago.

Upon visiting the skin practitioner or dermatologist, I asked him what the relationship between pruritus ani and the id manifestation of epidermophytosis was. I got a negative answer. Most men evaded me. In this report at Richmond, Va., we brought out the fact that 6 per cent of pruritus ani was of mitotic origin. We also broke that 6 per cent group into a 100

per cent base and alluded to the fact that only a fair percentage had a primary invasion of the perianal skin. That there was an id manifestation in a fair percentage of these individuals, an allergic manifestation, was also brought to light.

May I reiterate that id manifestation in the light of the dermatologist is a secondary eruptic or non-eruptive manifestation away from the primary focus of infection. That same type of phenomenon can be present about the perianal tissues from a primary on the feet, between the toes or the planar surface.

An allusion has been made to the fact that this treatment must be considered about the perianal tissues. No mention has been made in this general discussion of the importance of taking patients' shoes and stockings off and examining their feet along with the perianal examination. That is important.

Dr. Robert Terrell has alluded to the fact of the importance of perianal hygiene. May I reiterate that if you do not teach your patients how to cleanse the perianal tissues and keep them adequately clean at all times, literally being told they must bathe themselves after each evacuation for the remainder of their days, you are not arriving at one of the primary problems of treatment of prutitus ani.

Finally, and not least, the importance of eradicating distant foci of infection or primary focus of infection in other parts of the body, whether it be of an epidermophyton phenomenon or other allied skin problems, should be borne in mind.

Frank M. Frankfeldt (closing): As regards Dr. Guess' discussion, the results you are going to obtain in the interpretation and treatment of pruritus ani will depend on the thoroughness of your investigation, your knowledge of allergy and appropriate therapy if you have not found the incriminating allergin. There is no question about it and there is no fact to dispute the premise that if you find a pruritic skin there must be two factors: There must be a sensitization of the skin on the one hand and some irritant on the other hand. Otherwise, how can you explain the fact that every one of us sees thousands of patients with anorectal pathologic conditions when the skin is bathed with these irritating discharges and they do not itch. Well, why is it that other people do itch? There must be a rationalization for it. As ye seek, so shall ye find.

As busy proctologists, it is unfortunately much easier to take a Flit gun and shoot something under the skin than to spend a few hours investigating a case and treating the cause rather than the effect.

As regards fungus infection, I think no one has preached fungus infection more than I have. I think the members of the New York Proctologic Society will remember that quite a number of years ago I tried to emphasize the frequency of fungus infection as an etiologic factor in pruritus ani. In 1938, I had the privilege of collaborating with Sir Albert Castellani and we discussed the subject in detail. You do not frequently recover the epidermophyton either from the original focus of infection between the toes or from the id. Because a patient has athlete's foot and has pruritus ani, it does not necessarily follow that that patient has an id phenomenon; if you were to take the shoes and stockings off most of us here, we would be very much surprised how many people do have desquamation and evidences of infection between their toes. There must be a criterion. If a man like Castellani had examined one patient nineteen times before he could isolate the trichophyton or epidermophyton, how futile would be our attempts to try to get these organisms? Why have I included some of these patients under the caption "fungus allergy"?

Incidentally, when you get pruritus ani as a result of a fungus infection, you have a fungus allergy and you have a contact dermatitis which is caused by the products of metabolism of these fungi in the diseased skin, just the same as the other phenomena are allergy but, unfortunately, not understood. Now, to say a case is due to fungus, of course, the ideal thing is to be able to say, "I have found the fungus," which very few of us can say.

I know the work the elder Dr. Terrell did because I was in correspondence with him a number of years ago and at that time I was very much interested in fungus infection. Unless you have an acute exacerbation of an interdigital infection and then a perianal lesion, it is hard to say that that particular thing is due to a fungus infection.

It is hard to say that a perianal lesion is due to a fungus infection. If you had a tinea cruris and a pruritus ani, you would be more safe and more apt to be just in saying that that is a fungus infection because it was carried by a towel.

Gentlemen, there is one other point if I may make it. If you ever have an intractible patient, one who comes to you with agonizing itching, there is one thing that will stop the itching and you can bet your bottom dollar on it; that is ice. If you will take a throat ice bag and put it on a suspensory belt under the perineum, you will find that because of the contraction of the capillaries plus the freezing of the nerve ends, you will in almost every case be able to control the itching. If I had time, I could go into many more points.

Rachelle Seletz (closing): I want to thank the discussors for their very gracious comments. Everyone was right and everyone was wrong, of course.

In regard to the so-called id reaction of Dr. Hill, we all agree about the fungus; I mean most of us do. All of us seem to agree about incidence of fungus. It is right that we should look at the feet and look behind the ears or in the external auditory meatus or, if you please, even in the umbilical orifice because that is the usual sites for hiding away of fungi. Incidentally, it is not because we do not wash there but because we do not dry there.

When a person has interdigital epidermophytosis, it does not mean he particularly has an id reaction but it may mean that he is a vulnerable subject for fungi.

During the war many medical officers spoke about the incidence of fungi. A particular major with the combat forces described thousands of patients that he supervised, with only one instance of pruritus ani and that only in a soldier who did not want to be an artilleryman.

When you have the proper circulation, you do not get pruritus ani. This brings us back to our syndrome. One thing alone is not enough. The body mechanisms are not that simple. It is only when the proper neurovegetative imbalance is produced by preferably anal spasm, psychiatric factors with the hyperactivity of the perianal sweat glands, (as we discussed before), plus an overconsumption of carbohydrates, that the fungi have a chance to take hold of the skin and we will have pruritus as a result. It takes more than one contributing factor. You must consider pruritus ani as a syndrome to include all these separate factors.

MODERN SURGICAL TREATMENT OF HEMORRHOIDS AND A NEW RECTOPLASTY

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THE objectives to be sought in performing an operation for hemorrhoids are: first, to remove all pathologic tissue second, to preserve all the normal tissue third, to maintain or restore the normal anatomic configuration and thus to maintain or restore normal function fourth, prevent sequelae and complications and fifth, keep pain and morbidity minimal.

It is well to recall the special anatomy and physiology of these parts, the characteristics of the tissues and their type of healing when injured. The anal canal is an elastic, distensible and contractile tube surrounded in virtually its entire length by voluntary muscle with a sphincteric action, and in its upper two-thirds its wall also contains the involuntary sphincter. The lower portion of the lining is of ectodermal origin and in reality is modified non-secreting skin; the upper portion and the rectum proper is lined by mucous membrane which is of entodermal origin and secretes mucus. The line of junction of mucous membrane and modified skin is at the semilunar valves; it is 2 to 3 cm. from the anal margin. While connective tissue and epithelium regenerate, injured muscle does not regenerate but is replaced by relatively inelastic, non-contractile, fibrous tissue.

Stenosis, seepage and early recurrence of hemorrhoids are among the sequelae following hemorrhoidectomy. Their pathogenesis, briefly stated, herein follows: The stenosis may represent an actual narrowing or a loss in elasticity or distensibility of the tissues. The factors responsible may be the removal of too much circumference, damage to muscle and replacement by fibrous tissue and increased fibrosis as a result of infection. Seepage may be due to a downward displacement of the secreting tissue, namely, the mucous membrane, formation of inelastic bands or segments which do not permit adequate closure of the outlet, damage to the voluntary muscle with varying degrees of incontinence, damage to the involuntary sphincter and incomplete healing. The early recurrence of hemorrhoids is most likely due to inadequate removal at operation.

Any presentation on hemorrhoids should carry a note of warning that adequate diagnostic procedures be employed to rule out the presence of neoplasms and other disease before treatment is instituted. This includes a complete history, a careful digital and a thorough sigmoidoscopic examination after adequate preparation of the patient. Roentgenoscopy is reserved for the study of the parts above the reach of the sigmoidoscope.

The preoperative preparation includes phthalysulfathiazole (sulfathalidine) begun about four days prior to operation in doses of 1 Gm. four to five times daily and continued for seven to ten days after operation. The use of this drug seems to be followed by fewer instances of abdominal distention and by more that have spontaneous defecations than is seen when mineral oil or bulk laxatives are used. Furthermore it may reduce the tendency to fibrosis due to low-grade postoperative infection.

A bland diet is ordered twenty-four hours preoperatively to reduce the fecal content of the lower bowel so it may be cleaned out adequately by enema the evening before operation, and also to minimize abdominal discomfort following operation.

Sedation is obtained by the administration of sodium pentobarbital gr. 1½ the evening before operation; if local or re-

gional anesthesia is employed, this drug is repeated one hour prior to the scheduled time of surgery; in addition, morphine sulfate gr. $\frac{1}{6}$ and scopolamine in an ampule gr. $\frac{1}{150}$ to $\frac{1}{200}$ is administered hypodermically one-half hour before operation.

in hypertensive patients. Other cardiovascular stimulants have never been found necessary, the blood pressure changes varying in either direction not more than 10 mm. of mercury from the pre-anesthetic reading.

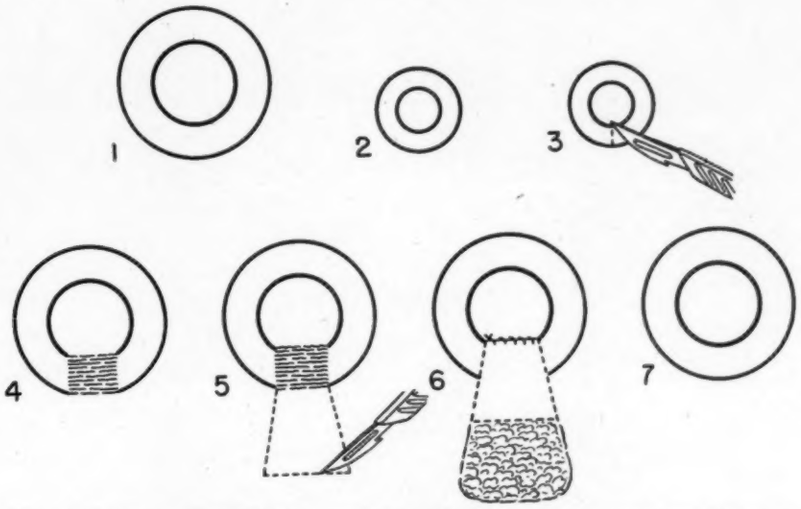

Fig. 1. Diagram illustrating preparation of sliding graft and anusotomy in plastic repair of anal stenosis, and also employed to prevent contractures following radical hemorrhoidectomies. 1, Represents normal anal lumen; 2, a stenosed canal; 3, indicates the anusotomy; 4, shows the separation of the wound edges and consequent widening of the anal tube; 5, represents the incisions for preparing the sliding skin graft; in 6, the graft has been drawn up to cover the entire surface of the anal wound except for two narrow radiating slits; a large open wound remains on the outside far from the margin; 7, indicates the end result.

Complete physical examination, urinalysis, complete blood cell count, hemoglobin and blood clotting time determination are part of the routine.

Spinal anesthesia in a much smaller dose than is customarily used has been employed by me over a period of fourteen years. Twenty mg. of procaine (1 cc. of 2 per cent solution) in a commercially prepared ampule, which also contains suprarenin 1:20,000, has been found to afford anesthesia for a period of one hour or longer. The omission of suprarenin may shorten the duration of anesthesia markedly. The small dose of procaine subarachnoidally seems to cause less strain on the patient's tissues generally and locally than other anesthetic methods and dosage heretofore used. The amount of suprarenin is so small that it apparently need not be feared

The time-honored lithotomy position has been replaced largely by the Buie prone position with buttocks elevated, and by either the left or right lateral position.

Reagents which produce prolonged anesthesia definitely have been established to result in less postoperative pain, which is evidenced not only by the smaller number of doses of the narcotic employed but also by the calmer attitude of the patient. The oil-soluble reagents and diothane solution 0.5 per cent have been found to be satisfactory. Yet even more important than these in the reduction of postoperative pain is judicious, adequate and gentle surgery for which there is no substitute.

The objectives mentioned at the outset will be secured if the surgeon restores or retains the anatomic features, namely, a canaly of sufficient caliber to admit readily

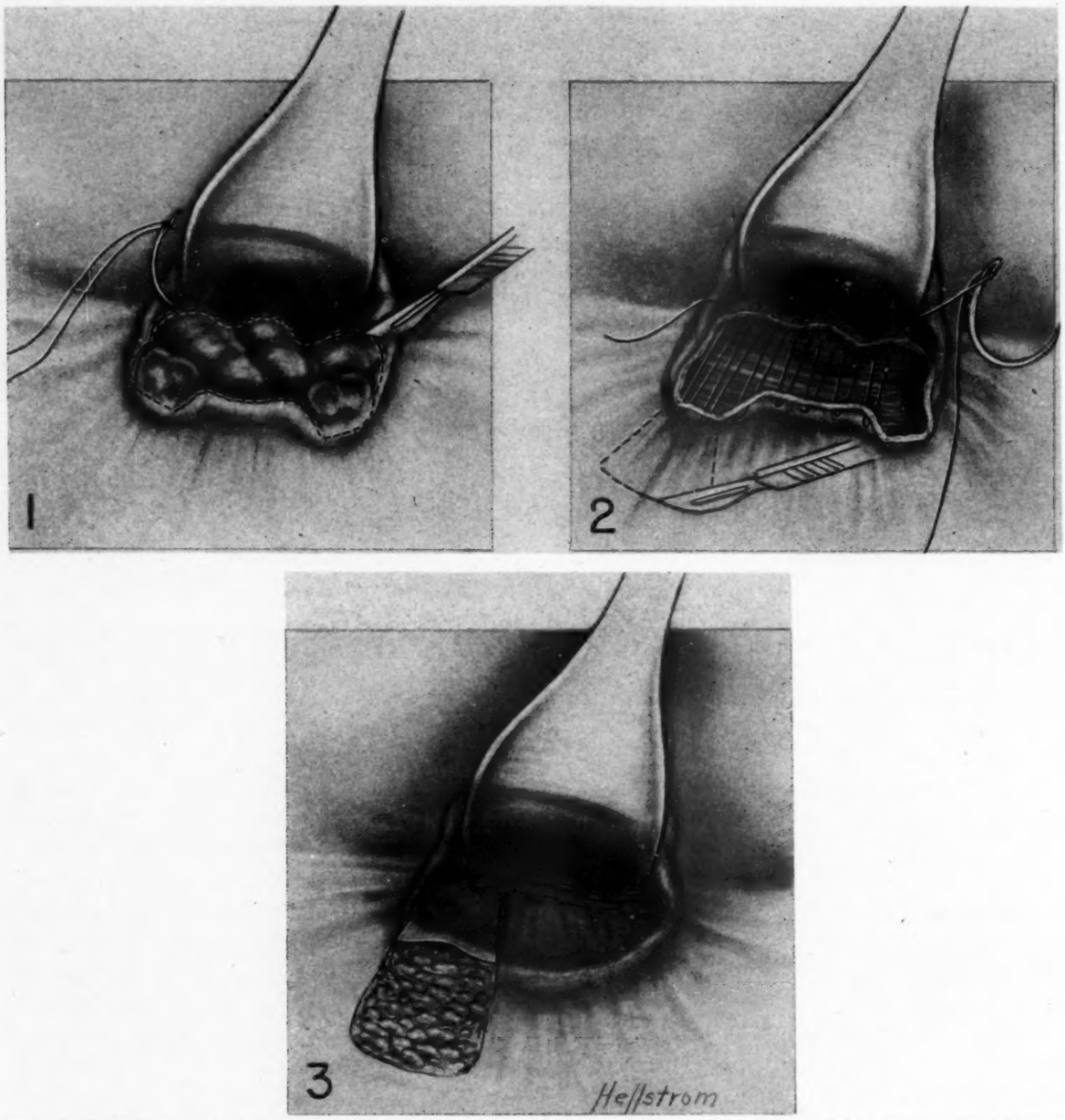

FIG. 2. Radical type of circumferential hemorrhoidectomy, with utilization of sliding graft. In 1, three initial transfixion sutures are shown; the dotted lines indicate the lines of excision on one hemicircumference; 2, depicts the outline of a sliding graft and the sutures employed for loosely coapting the wound edges; in 3, the graft has been drawn up into the anal canal, and the suturing completed; the wound edges in the right side of the illustration could easily be united without producing tension, hence no additional graft is shown.

at least two fingers with the patient under anesthesia, walls that are elastic, distensible and contractile and a mucocutaneous junction which is no lower than the normal pectinate line, that is about 2 to 3 cm. from the anal margin.

In general, two surgical methods are now in use, namely, the radial excision method and the circumferential excision or amputation method.

Radial excision procedures are apparently the most frequently employed and it has been emphasized—and rightly so—that adequate islands of uninjured tissue should be left lest stenosis ensue. Many such technics have been described which are

satisfactory for patients with simple cases involving scarcely more than the three classical sites of the tube, namely, the left lateral, the right posterior and the right anterior segments.

The patients with extensive cases still posed a problem for the surgeon studying anorectal diseases. If all the involved tissues are simply removed either radially or by amputation and suture of the wounds, the undesirable sequelae mentioned are prone to follow. On the other hand, if all the abnormal tissue is not removed, as has been frequently advocated in order to avoid sequelae, the result again is apt to be unsatisfactory, in that the patient's symptoms may not be eradicated completely or early recurrence may ensue.

Contributions toward the solution of the dilemma include the creation of anal flaps in surgery of prolapsed strangulated hemorrhoids,[1] suturing of the superior cut edge of the mucous membrane to the proximal fibers of the external sphincter muscle[2,3] and creation of three or four isthmuses by suturing the cut mucous membrane to the corresponding cut skin and employing avaliable external tags to aid in forming the isthmus while allowing the rest of the wounds to granulate.[4]

It is generally conceded that tissues sutured properly without tension heal more satisfactorily than raw granulating surfaces. I have found it quite advantageous to employ sliding skin grafts and relaxation incisions and occasionally anusotomy to aid in filling defects of the anal canal and to prevent contractures. The end result tends to be a tube which simulates the normal hitherto unoperated one. The method was first used in 1936 in correcting a marked anal stenosis (Fig. 1), in patient H. L. at Longview Hospital, and was orally described by me at a round table discussion at a meeting of the Ohio State Medical Association in Cincinnati, 1940.

After the hemorrhoidal tissues have been removed a graft is prepared by making an incision in the perianal skin about 1.5 to 2 cm. lateral to one or more of the wounds made by the dissection of the external hemorrhoids. At each end of this incision a radiating cut is made, thus creating a segment of skin detached at all its edges and attached only by its deep surface and capable of being slid without tension well up into the anal canal to the level of the cut edge of the mucous membrane. (Fig. 2.) The medial edge of this skin graft is sutured to the superior portion of the external sphincter muscle and to the mucous membrane which is not pulled down but rather the skin is pulled up to it. Thus, the anal canal becomes relined by tissue of ectodermal origin and the mucocutaneous border is reestablished at the proper level. The radiating cuts not only help to free the sliding graft but serve as drainage slits. Sometimes it is found that the incision which is made lateral to the anal margin releases the tension sufficiently to allow the skin to be pulled up to cover the defect completely, making it unnecessary to add the radiating cuts; this constitutes a relaxation incision. One or two—rarely three—such incisions or grafts are employed.

The addition of relaxation incisions and sliding grafts to any method of hemorrhoidectomy permits the removal of all diseased tissue and the final results of such a plastic repair frequently challenge detection of any operation having been performed or of disease having existed.

COMMENTS AND SUMMARY

1. The objective of modern hemorrhoidectomy is the complete restoration to normal by the removal of all diseased tissue and the application of plastic methods of repair when indicated.

2. Before treatment is instituted the presence of neoplasms and other disease is ruled out by a complete proctologic examination including a thorough sigmoidoscopy; x-ray is employed for the detection of lesions above the level that can be reached by the sigmoidoscope.

3. Phthalysulfathiazole (sulfathalidine) seems to add to the postoperative comfort of the patient by lessening abdominal dis-

tention and by promoting spontaneous defecations.

4. Spinal anesthesia, with a much smaller dose of anesthetic reagent than is customarily used, is advocated. One cc. of a 2 per cent procaine solution containing suprarenin 1:20,000 (20 mg. of procaine) has been found to be effective and innocuous in both hypertensive and hypotensive patients. The use of suprarenin in the procaine solution doubles or may even quadruple the duration of anesthesia.

5. For the reduction of postoperative pain the most important factor is good surgery; reagents which produce prolonged anesthesia are of value.

6. True sliding grafts and relaxation incisions in conjunction with hemorrhoidectomy are described. Their use permits radical removal of hemorrhoids and restoration to normal without sequelae.

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DISCUSSION

Hugh Beaton (Fort Worth, Tex.): Someone has said, "The most successful man is the man who holds onto the old just so long as it is good, and grabs the new just as soon as it is better." Dr. Carmel has done that. Certainly we all agree with him about the value of a thorough and complete physical examination. Embarrassing situations can be avoided by knowing the patient's physical condition before starting an operation. I have not used sulfathaladine in preoperative care. I think I probably will in some of my patients afterwards. My patients are in the hospital the afternoon before the morning of operation, in ample time for routine laboratory work followed by a good night's rest.

For anesthesia I prefer caudal, metycaine 25 to 30 cc. However, at times nitrous oxide and ether are used; also, spinal, 50 mg. (I have not been able to get along with less.) In the aged, I find local infiltration very satisfactory. In some cases I use oil-soluble anesthetics but it is my impression that they are not without danger and I believe they retard healing in some patients.

Regarding surgical technic so ably described by Dr. Carmel, I can see the work of Drs. Buie, Bacon, Fansler, Hirschman, Martin, Rosser, etc.

I have done many plastic type operations and, although using careful technic and postoperative care, every now and then I get an anal stricture which has to be taken care of in the office. I would like the Dr. to tell us if he has the same difficulty.

What we are striving for in rectal surgery is to remove all pathology, avoid unnecessary trauma and leave a clean, flat wound that heals readily. When the first dressing is removed the, anus should be free from edema and tags. At the close of operation I apply mercurochrome and a small wick of oxycel held by a T-binder.

Patients are allowed out of bed to void and are given sitz baths the following day. I do not apply warm packs unless the patient has special nurses. I keep my patients in the hospital on an average of seven days. They come to the office once a week generally for four weeks. By that time the wounds are healed; the anal canal is normal, at least it is normal so the patient can have a well formed bowel movement without pain.

I wish to thank Dr. Carmel for the privilege of discussing his paper.

A. Gerson Carmel (closing): I want to thank Dr. Beaton for his remarks. The application of the principles and procedures I mentioned affords the experienced proctologic surgeon an increased latitude in excising all pathologic tissues.

SURGICAL TREATMENT OF CHRONIC ULCERATIVE COLITIS

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THOSE who have been following the management of chronic ulcerative colitis over the past decade or longer are keenly aware of the controversial nature of this disease.

In reviewing the records and ultimate outcome in 110 patients with chronic ulcerative colitis seen in the twelve-year period from 1935 to 1946 I am favorably impressed by those who have been classified as mild. They have continued in fair health in their respective occupations and satisfactory management has been evolved by their physician.

However, I am concerned with a group that has been classified as advanced cases of chronic ulcerative colitis. It is probable that about 15 to 20 per cent of all patients with chronic ulcerative colitis reach an advanced stage. Whether this follows an acute fulminating onset or is a result of recurrent attacks is of some importance and will be considered later.

If there is some degree of accuracy in the estimate that 15 to 20 per cent of all patients with chronic ulcerative colitis reach an advanced stage, we are then ready to discuss treatment of this group of patients.

If we are to evaluate properly both medical and surgical treatment of this group of desperately ill patients, some rules of conduct should be formulated.

It would seem wise to state that the objective of medical and surgical treatment should be "a restoration to health and previous occupation." This should also be the yardstick by which we measure the results of both methods of treatment. Finally, if we have failed to rehabilitate the patient so that a "medical derelict" or a "surgical invalid" represents the outcome of our medical or surgical efforts, we should re-evaluate our methods of treatment.

DIAGNOSTIC CONSIDERATIONS

Our patients have been classified as having chronic ulcerative colitis when they presented clinical, roentgen and proctoscopic features of this disease as described by Rankin, Bargen and Buie.[1] Amebic colitis, tuberculous colitis, bacillary and other forms of colitis have been excluded by appropriate investigation.

It seems desirable to state that a definition of an advanced stage of ulcerative colitis will be found under each surgical heading in another section. However, it is to be noted that the advanced stage of ulcerative colitis was reached by some of these patients in two rather distinct ways: (1) As a result of an initial fulminating onset, some patients arrived at a rather severe state of constitutional decline and structural change in the colon. These patients all presented intricate problems in the management of their sepsis, anemia, malnutrition and diarrhea. I have not been impressed by the results of those who advocate ileostomy as a life saving procedure for patients in this phase of the disease. Whether or not these patients die under medical or surgical auspices is dependant upon who is taking care of them at the time. (2) Those patients who, as a result of chronic or recurrent ulcerative colitis, reached an advanced stage of constitutional and visceral degenerative changes, with or without complications, were easier to manage. A few of these patients died as a result of surgical treatment aimed at restoring them to health and previous occupation but the majority survived and were rehabilitated. I am impressed by the surgical rehabilitation of this group.

When any patient became a candidate for surgery as previously described, we then

classified the indications for surgery under the terms A—Specific and B—Elective. In so doing, the reports of Cave, Jones, Rankin, Cattell, Dixon, Bargen, Bockus, Crohn, Garlock, Kiefer and many others were followed with interest and profit.

SPECIFIC INDICATIONS FOR SURGERY

Chronic Ulcerative Colitis with Constitutional and Visceral Degenerative Changes. The typical patient in this group has had either a chronic or intermittant ulcerative colitis for several months or years. The clinical features of chronic sepsis, toxicity, anemia, hypoproteinemia, loss of weight and strength and signs of multiple vitamin deficiencies will be present. In young women cessation of menses, failure of breast development and other secondary sex characteristics may be noted. In both sexes there have been numerous prolonged periods of absence from school or work. The patient is a chronic invalid and a candidate for surgery.

Anorectal Complications. With the development of stricture, recurrent abscesses, anorectal, pelvic and rectovaginal fistula, incontinence and painful disabling perirectal infection, an insurmountable barrier to recovery has been reached. Adequate drainage of abscesses should be done but beyond this further local surgical treatment is usually unsatisfactory. The disability suffered by the majority of patients in this group prevents them from enjoying good health or full employment. They are candidates for surgery.

Polypoid Degeneration and Carcinoma. There is almost universal agreement that one should consider malignant change as the usual outcome of polyp formation, regardless of whether these polyps are considered inflammatory pseudopolyps or true adenomas. Polypoid degeneration ordinarily represents a complication of long standing chronic ulcerative colitis and diagnosis can be made by proctoscopy and careful roentgen examination. Good surgical judgment indicates that these patients are candidates for operation despite the fact that disability may be minimal in some instances.

Obstruction and Tumor Mass. An abdominal or abdominopelvic mass arising from any portion of the colon may produce obstruction. The diagnostic problem of determining whether the tumor mass is inflammatory or malignant can rarely be solved without exploration. A previous roentgen or proctoscopic diagnosis of polypoid degeneration may favor the diagnosis of malignancy. Relief of the obstruction by appropriate surgery is indicated preliminary to surgical treatment of the mass itself.

Subacute Perforation, Abscess and Fistula. With the development of an abscess resulting from perforation it is advisable to perform drainage after localization has occurred. Surgical experience indicates that the improved condition of the patient following such drainage enhances the success of subsequent surgical procedures. With the development of a fistula ileostomy or proximal colostomy should be done several weeks or months before any resection of the involved portion of the bowel is attempted.

ELECTIVE INDICATION FOR SURGERY

Focal Infection. Some individuals, as a part of their chronic ulcerative colitis picture, develop a disabling polyarthritis. It is conceded that they may be benefited by resection if the arthritis has not advanced to the stage of marked structural change in the joints, tendons, synovia, etc. Pyodermia, recurrent skin ulcers, furunculosis, neuritis, stomatitis and erythema nodosum are some of the manifestations of this disease that may be avoided or benefited by eliminating the source of infection.

Hemorrhage. I do not find myself in accord with the heroic viewpoint that chronic blood loss from recurrent or continuous hemorrhage is an indication for ileostomy. Massive hemorrhage has responded to repeated transfusions of blood, plasma and protein solutions. Massive parenteral doses of vitamin K, C, niacin, B complex and liver are part of the sup-

portive treatment. If the patient can be carried over this critical stage, a high surgical mortality rate will be avoided and chances of a favorable outcome will be increased.

It is acknowledged that some patients will die from exsanguinating hemorrhage and infection because they neither respond to supportive treatment nor become acceptable for surgical intervention.

Acute Fulminating Ulcerative Colitis. Surgical treatment should be avoided in the majority of patients who develop initial acute fulminating ulcerative colitis. An initial fulminating attack of ulcerative colitis developing in a patient who has heretofore been well should be considered a medical problem. These patients develop an advanced structural pathologic change in their colon very rapidly and the severe state of constitutional decline is quite alarming. If these patients can be carried over their initial fulminating attack, some will enter a relatively afebrile ambulatory phase and respond to medical management.

However, if they show signs of returning to a toxic febrile phase when they have apparently recovered from their initial fulminating attack, surgical intervention is advocated.

Acute Perforation. With rare exception perforation into the free peritoneal cavity during the initial acute stage of ulcerative colitis is fatal. A section of necrotic colon full of holes and the inability of the adjacent bowel, mesentery and viscera to cope with such a severe spreading peritonitis as may be demonstrated at the autopsy table is ample evidence of the futility of treatment.

SURGICAL PROCEDURE

A few general remarks concerning the object of surgical treatment are in order. If we remember that our goal is surgical rehabilitation of the patient, we must define this as a return to previous occupation and good health without limitation of activities. If we are to rehabilitate the patient, morbidity and mortality should be kept to a minimum or we have not fulfilled the object of surgical treatment. Finally, if we have succeeded in rehabilitating the majority of these patients, our mission has been fulfilled.

The surgical procedures have been planned to suit the requirements of the patient.

Ileostomy without Colectomy. Ileostomy without colectomy was performed for six patients. Four have been restored to health. One patient died the fifth postoperative day and must be considered in the postoperative mortality figures. One patient survived ileostomy but subsequent to his discharge home developed an ileocolic fistula and died as a result. This death should be attributed to the disease and should not be considered in the operative mortality figures.

Ileostomy—Total Colectomy—Rectum Left in. Two patients have had a total colectomy performed in one stage. Ileostomy had preceded the colectomy and the rectum was left in. In one patient the rectum became involved after total colectomy was performed and in the other the rectum was involved at the time colectomy was performed. Both will have an abdominoperineal resection at a later date although their restoration to health has been accomplished. The third patient had an unsuccessful ileosigmoidostomy converted into a permanent ileostomy due to recurrent anorectal abscess-fistulas. He is employed full time.

Ileostomy—Total Colectomy—Abdominoperineal Resection. Three patients have had ileostomy, total colectomy and abdominoperineal resection of the rectum done in stages. All have survived and are gainfully employed without loss of time or disability.

Ileostomy—Right Colectomy. One patient had ileostomy and right colectomy done at the same time. She died the tenth postoperative day from perforation of the colon.

Left Colectomy—Abdominoperineal Resection. Four patients who had an ulcerative colitis involving only the rectum and

descending colon have been restored to their previous occupations. They have a terminal colostomy of the right transverse colon and are not disabled.

Ileosigmoidostomy—Total Colectomy. One patient had a successful ileosigmoidostomy followed by total colectomy performed in two stages. During her convalescent period at home she developed symptoms of incomplete obstruction due to adhesions. She was re-admitted and died following exploratory laparotomy. Her death is considered a postoperative hospital mortality in the statistics.

Segmental Resection—Anastomosis. One patient had resection of the descending colon for a segmental form of chronic ulcerative colitis. End-to-end anastomosis between the transverse and sigmoid colon was performed. She is gainfully employed.

Colostomy—Inoperable Carcinoma. One patient had a colostomy performed in the right transverse colon to relieve obstruction. This patient subsequently died in the hospital from carcinoma of the sigmoid arising in a segment of ulcerative colitis. Her death is correctly attributed to the disease and not to the operative procedure.

ANALYSIS OF SURGERY

It is difficult to analyze the status of the operative procedures for in tabulating these it was found that twenty patients underwent thirty-five operations. A postoperative mortality of 8.5 per cent due to the operative procedures is acknowledged as correct. Following operative procedures a mortality of 5.7 per cent due to the disease, but not the operative procedure, is likewise correct.

In commenting on Table I it is to be noted that under numerals II, III and V the rather formidable procedures of subtotal or total colectomy with or without abdominoperineal resection of the rectum were done without a fatality. This is indeed a fortunate and happy experience for I have developed a distinct feeling that if I can get my patients over the ileostomy stage of their operation the rest would be easier.

In commenting on Table II a restoration to health and previous occupation was accomplished in 70 per cent of patients. This is indeed gratifying for it should be obvious that rehabilitation of such a seriously ill group of individuals is a great problem.

In commenting on Table III I would like to point out that a direct relationship be-

TABLE I
STATUS OF OPERATIVE PROCEDURE IN TWENTY PATIENTS

35	Operative Procedures	Deaths	
A	Postoperative Mortality	No.	Per Cent
I	6 Ileostomies without colectomy	1	16.6
II	3 Ileostomies—total colectomy—rectum left in	0	
III	3 Ileostomies—total colectomy—abdominoperineal	0	
IV	1 Ileostomy—rt. colectomy	1	100
V	4 Left colectomy—abdominoperineal	0	
VI	1 Total colectomy—ileosigmoidostomy	1	100
VII	1 Segmental resection—anastomosis	0	
		3	8.5
B	Chronic Ulcerative Colitis Mortality		
	1 Ileostomy—ileocolic fistula (from 1 above)	1	100
	1 Colostomy—carcinoma—inoperable	1	100
		2	5.7

TABLE II
STATUS IN RELATION TO OBJECTIVE IN TWENTY PATIENTS

		Per Cent	
12	Restored to health	60	70
2	Convalescent	10	
3	Postoperative mortality	15	25
2	Chronic ulcerative colitis	10	
1	Unknown	5	5
20		100	

tween operative procedure and cause of death is present in three patients. However, in two patients death could not be attributed to operative intervention by any stretch of the imagination. One patient formed an ileocolic fistula during his convalescent period. This effectively voided the previous ileostomy and his death is correctly attributed to an active resumption of ulcerative colitis. The other patient died from carcinoma of the sigmoid and colostomy was performed only as a palliative procedure.

TABLE III
MORTALITY STATUS IN FIVE PATIENTS

A	Postoperative Mortality	15 per cent
1	Ileostomy—perf. ilium—peritonitis	
1	Ileostomy—rt. colectomy—perf. colon—peritonitis	
1	Explore—adhesions—peritonitis	
B	Chronic Ulcerative Colitis Mortality	10 per cent
1	Ileostomy—ileocolic fistula	
1	Colostomy—carcinoma—inoperable	

SUMMARY

1. In our limited experience it is believed that approximately 15 to 20 per cent of patients who have chronic ulcerative colitis will become candidates for surgery.

2. These patients will be advanced cases of ulcerative colitis as a result of either an acute fulminating phase or a chronic intermittant process.

3. A group of patients with advanced chronic ulcerative colitis were classified as candidates for surgical treatment under two headings: A—Specific and B—Elective.

4. Operative procedures have been briefly outlined.

5. A restoration to health and previous occupation, the objective of surgical treatment, was accomplished for 70 per cent of the patients.

DISCUSSION

ROBERT A. SCARBOROUGH (San Francisco, Calif.): Dr. Ault's paper, to which you have just listened, is a splendid exemplification of the type of critical analysis of one's own experience which all of us should periodically employ for our own good and for the good of our patients.

Dr. Ault has reason to be pleased and satisfied with the results of his surgical management of this disease. He has expressed no such pleasure or satisfaction, however, but rather encourages discussion of his failures.

There were five deaths in twenty patients. There was one death following ileostomy. This much comment is made only to emphasize that the performance of ileostomy for chronic ulcerative colitis is a serious major surgical procedure which can be followed by extremely high mortality unless the time of operation is wisely selected and unless preoperative and postoperative care are suitably controlled. Dr. Ault's mortality rate following ileostomy, which is actually 8⅓ per cent and not 16⅔ per cent, as shown in his own tables, is well below the average mortality rate for this procedure.

One patient died following simultaneous ileostomy and right colectomy. We believe that ileostomy alone should be a first stage procedure prior to any subsequent resection for chronic ulcerative colitis.

One patient died of peritonitis following ileosigmoidostomy and colectomy. This case would appear to exemplify the hazards of complications by infection in the performance of anastomosis in a colon involved by this disease.

The fourth death followed the development of an ileocolic fistula subsequent to ileostomy. This was an unforeseen and unavoidable complication which might have been prevented by early colectomy if it could have been anticipated.

The final death from inoperable carcinoma superimposed on chronic ulcerative colitis is of significance as an example of the potentiality for malignant degeneration in this disease. The time to cure the patient had passed when he first came to Dr. Ault. Only colectomy before malignancy developed would have saved this individual.

In our own experience, one of the most difficult decisions to be made has been that of just when to perform ileostomy. I agree with

Dr. Ault that it is not indicated in acute fulminating ulcerative colitis or for the treatment of hemorrhage.

It is indicated in those cases of chronic disabiling disease which have failed to respond satisfactorily to medical treatment.

Subsequent colectomy should be employed when septicemic toxic effects persist or when there is evidence of the development of polypoid mucosal proliferation, since this is a definite precursor to malignant degeneration.

Vernon G. Jeurink (Denver, Colo.): Not many years ago, we frequently heard the statement made that chronic ulcerative colitis was never a surgical disease, even when complicated by various conditions which now almost universally are considered indications for surgical interference.

Dr. Ault's presentation and Dr. Scarborough's discussion should impress us, along with an increasing volume of similar reports by other workers in this field, that surgery has a great deal to offer in the selected cases of chronic ulcerative colitis. Because of our personal inclination to leave well enough alone, we have been inclined to let our patients go along with ileostomy so long as their condition remains stationary or improved. When these conditions do not exist, or when stenosis, polypoid change and related changes occur, we recommend colectomy.

Recently, in ten cases only we have tried a new drug called nisulfazole which is still in the investigative phase. This is obviously not a sufficient number of cases from which to draw any worth while conclusions, but it is our impression that it may prove to be of some value in quieting acute exacerbation of the condition.

It has been a pleasure to hear Dr. Ault's paper and Dr. Scarborough's discussion.

Thomas T. Mackie (Winston-Salem, N. C.): It is a privilege to have the opportunity of discussing a paper of this sort. I, of course, am not a surgeon and I must confess that I am cheered to find a surgeon who has such a conservative point of view with respect to this very distressing disease. I agree completely with Dr. Ault's philosophy. I would add only one additional indication in his specific group and that is the rare case in which the extensive pathological development of this disease is restricted to the proximal colon. In those instances, I believe one should consider surgery at a very much earlier date than we do in the other types.

I agree completely that surgical intervention in the elective group is almost invariably followed by serious results and very seldom fulfills the objective which Dr. Ault has defined as the purpose of surgery, namely, the restoration of the individual to health and to a productive life. He has brought out also by indirection one of the other extremely important aspects of this problem which has impressed me over the time that I have been studying it. That is that there are very few of us who have sufficient breadth of experience to be fully competent to evaluate all of the problems which the individual case will present and that in effect many of these patients, and particularly the seriously ill ones, are conjointly medical and surgical problems, and that the best results occur when the internist and the surgeon are working together.

Again, I should like to congratulate Dr. Ault on a very interesting paper and, obviously, on very skillful surgery.

Frank C. Yeomans (Great Neck, N. Y.): I have no prepared discussion but I want to express my appreciation to the essayist and at the same time to emphasize again, of course, that chronic ulcerative colitis is essentially a medical disease. It stays medical until pathologic complications supervene. I have seen a number of cases of ileostomy and most of those patients are far from happy.

I want to call attention again to a surgical procedure that is now very much neglected. It was used a long time ago in cases of chronic ulcerative colitis that do not respond to treatment and are not complicated, we will say, by polypi. Appendicostomy is a simple and safe procedure and may result in great satisfaction.

At the meeting of the New York State Society in 1946, I showed a patient and reported on a man—I think he was fifty-six years of age—who was in a very desperate condition with chronic ulcerative colitis. He had been ill for months and received all the surgical treatments, including chemotherapy, transfusions and all that in a very good hospital and under a man who is very competent and has a large experience. He was given up by the doctor and told that he could either stay in the hospital and die or go home and die. So he was brought to the Polyclinic Hospital in an ambulance. He weighed about 90 pounds, had a temperature of 103°F., and was anemic,

lethargic, and seemed to be a candidate for the mortuary.

We tried some more medical treatment which failed. Then I did an appendicostomy, and through this opening irrigated the colon and instilled sulfa drug in suspension. In three days the picture changed. The ulceration healed completely and the man has remained well since a year ago last October. He still has the appendicostomy.

The advantage of appendicostomy is that if you have to do major surgery later, the appendecostomy does not interfere with it at all. It may itself be the life-saving procedure. If it is properly done, only a very small catheter, say a No. 12 French, is inserted and removed after each treatment. The patient learns to pass it himself. It is no inconvenience at all. There is no bag worn and it has all the advantages of a simple thing. It must not be closed. The mucosa grows to the skin; the stoma does not contract. It should not be closed because you do not know when it may be needed at a subsequent time. I have had patients who have had the appendicostomy for years and have had to use it on various occasions.

I bring that up simply to show that that procedure can be performed under local anesthesia or a little pentothal. It is not a major procedure comparatively and I bring it to your attention as a very useful and at times a life-saving procedure of a minor nature compared to the other.

Garnet W. Ault (closing): As I left the platform, several people asked the name of that bag. I will put it on the blackboard for you—*Rutzen*. I will have two of these bags on the table after the meeting so that you can examine them.

I wish to thank all discussers for their pleasant comments and am delighted to know we have some agreement and some very critical analysis. I would like to reiterate the fact that colostomy or ileosigmoidostomy should not be done in the presence of an involved segment of the bowel. I have not done that and I purposely made that plain and pointed out in one of those illustrations that the involved rectum would not stand the anastomosis of the ileum to it.

Dr. Scarborough has very definitely brought out the fact that polypoid degeneration will lead to carcinoma, and when your patient has reached that stage he is not a candidate for anything. We recognize that in the one patient in whom we did ileostomy, (as a part of a right colectomy) an error of surgical judgment was made. That is acknowledged.

I want to thank Dr. Jeurink for his discussion. We use the sulfas, streptomycin, penicillin and all other forms of chemotherapy as useful adjuvants.

I am delighted to hear that Dr. Mackie could be here and I appreciate his discussion. I am in thorough agreement that with lesions involving the proximal colon, we should do our surgery earlier. I am in complete agreement that these patients should have joint medical and surgical management. If you attempt to manage these patients alone from the surgical standpoint, you are headed for trouble. You should have joint management and you will have much better results.

I do wish to point out that 15 to 20 per cent of all cases of ulcerative colitis become problem cases. Whether they become problem cases due to an initial acute fulminating attack or due to chronic recurrent attacks is an entirely different thing; both require joint management. In the group of 15 to 20 per cent, who become difficult to manage, surgery has something to offer these patients who can be considered candidates for surgery.

I want to thank Dr. Yeoman's for his discussion. I would like to ask why are Dr. Yeomans' patients not happy with ileostomy? I am wondering if some people are not still using apparatuses that are not suitable for ileostomy. The Rutzen bag is the best answer to ileostomy. It is the only thing that we know that has made surgery acceptable to the physician and to the patients. If you are going to do an ileostomy and put an ill-fitting bag on, put sea sponges or something else on the surface, you are not going to have a happy patient and you are not going to have a very happy doctor very long, either.

CHRONIC DIARRHEAS*

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CHRONIC diarrhea is perhaps the most grossly mismanaged condition in all of medicine. The reasons are not far to seek: The first is that it is likely to be treated by home measures or drugstore advice until it has become established and until in some patients it is no longer simple. The second reason is clear from the first. It is regarded as a clinical entity and is treated as if it were whereas actually it is a symptom for which an underlying cause must be sought. At best the etiologic diagnosis is not easy, in that it is a laboratory matter which calls for special technics. At worst it is long and tedious and often unrewarding. There are, however, no short cuts and it is the attempt to take them which leads to so many difficulties in the diagnosis and treatment of diarrheal diseases.

The diarrheal diseases are with rare exceptions non-lethal but they represent an important problem in medicine today. They have a high incidence. They affect all groups and classes although their frequency increases as one descends the social and economic scale, their spread being favored by poor hygienic habits, which unfortunately go hand in hand with poverty. Their spread is also favored by the fact that many patients affected with them do not consult physicians. In their extensive survey of acute diarrheal diseases, for instance, Hardy and Watt found fecal cultures positive for shigella in 380 persons, of whom only two were under the care of a physician; one of the two died three days later. As these writers note, in the absence of their special study, there would have been 378 undetected infections and a corresponding unrecognized number of hidden sources of new infections.

Another reason for the spread of diarrheal disease is the frequent failure of the physician who is consulted to take the time and make the effort required to identify the etiologic agent. It is unfortunate that diarrhea usually responds rapidly, although often transiently, to simple measures. Under the circumstances it takes a certain strength of character on the part of both physician and patient to undertake a long routine of investigation.

Definition. A discussion of the chronic diarrheas properly begins with a definition of terms. Diarrhea must be distinguished from dysentery. The etiologic bases of the two symptoms are sometimes different and from the standpoint of severity they are always very far apart.

It is both useful and convenient to make the distinction in terms from the patient's point of view. The passage of unformed stools characterizes both diarrhea and dysentery. Diarrheic and dysenteric stools may both contain mucus, pus and blood but a dysenteric stool also contains cellular débris, it contains little fecal matter or none at all and its passage is always associated with tenesmus. A diarrheic stool, on the other hand, whether the diarrhea is acute or chronic, is chiefly fecal in composition.

From the patient's point of view diarrhea represents an abnormality in bowel habit as compared with the usual normal habit. It may manifest itself by an increase in the number of stools as well as by a change in their characteristics. What would be diarrhea to one person would not be at all ab-

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normal to a person whose normal habit was to pass two or more semiformed or soft stools daily. Detailed questioning, however, is always necessary for definitions of normal differ. One patient in the writer's experience who stated that her bowel function was normal, later revealed that for years she had had as many as eight bowel movements daily. A dysenteric stool, on the other hand, is not a matter for relative consideration. It would be considered abnormal by any patient, regardless of what his usual bowel habits were.

The classification of diarrhea into acute and chronic has nothing to do with the severity of symptoms but is entirely related to time. For the purposes of this discussion a chronic diarrhea is any diarrhea which is continuous or recurrent and which has lasted longer than a month.

ETIOLOGIC CONSIDERATIONS

On the basis of personal practice, which for the past eleven years has been confined exclusively to diseases of the colon, it may be said that amebiasis, shigellosis and brucellosis account for two-thirds to three-quarters of all patients with chronic diarrhea who are seen. Whether this is generally true is perhaps open to debate. The writer's practice is chiefly consultative and much of it comes from outside of Louisiana. It is doubtful, however, that his practice represents an accurate cross section of the diarrheal diseases in the United States and it is equally doubtful, in spite of a large number of patients referred from Central America, whether it represents an accurate cross section of these diseases in the tropics.

In the order of frequency, amebiasis is the most common cause of chronic diarrhea although if the physician relies upon diarrhea alone to make the diagnosis, he will miss more than one-half of all his patients. This is a disease in which constipation is an important part of the clinical picture.

The second most frequent cause of chronic diarrhea is shigellosis. In the writer's experience it far exceeds the number of patients with bacillary dysentery observed over a comparable period of time. Not more than 20 to 30 per cent of all patients with shigellosis, furthermore, can supply a history of previous dysentery although a carefully taken history very often reveals previous abdominal complaints. They may be little more than vague discomfort but they justify the assumption that one is dealing with a long-standing low grade infection.

Brucellosis, although its importance as a major epidemiologic problem in the United States is becoming clear, is still not frequently recognized in its intestinal form. In twenty-six patients recently seen in consultation, it had previously been suspected as the explanation of chronic diarrhea in only two instances. The other diagnoses included amebiasis, amebic dysentery, malaria, spastic and non-specific ulcerative colitis, cholecystitis and diarrhea of undetermined origin. The number of recognized cases of intestinal brucellosis is increasing, probably because the disease is being sought for. The writer's experience is to that effect. Over a five-year-period when no particular endeavor was being made to identify the disease, only nine cases were observed. The twenty-six patients just mentioned were seen over a two and one-half-year-period during which the possibility of intestinal brucellosis was increasingly borne in mind. At the present time five or six patients with this disease are being identified every month.

Another point to be emphasized in a consideration of the chronic diarrheas is the possible coexistence of two or even three different types of intestinal infection in the same subject. It has been known for a long time that amebiasis can coexist with shigellosis but personal experiences with returning servicemen from the Southwest Pacific have been particularly striking in this respect. Some of them have presented extremely obstinate amebic infections which did not respond to treatment based on sound concepts and which were ordinarily effective. In a number of cases further in-

vestigation revealed the reason, that a Shigella infection was present also, and therapeutic results were not achieved in amebiasis until shigellosis had been controlled with autogenous vaccine.

A similar situation may develop when amebiasis coexists with brucellosis. When it does, the amebiasis will be refractive even to adequate treatment until treatment for brucellosis is carried out. Indeed, refractive amebiasis, after adequate treatment, is sufficient indication for a search for brucellosis whether or not the disease is associated with intestinal manifestations. The writer's own practice is highly selective but it is probably suggestive of the general clinical picture so far as chronic diarrheas with dual etiologies are concerned.

With the exception of passing mention of one or two puzzling but relatively unimportant diarrheal diseases, this discussion will be confined to the three most common ones, amebiasis, shigellosis and brucellosis, and they will be discussed only from the standpoint of certain considerations which in the writer's experience have proved to be important. There will be no discussion of diarrhea due to giardiasis, which is not common. Schistosomiasis is not of significance in the United States and strongyloidiasis, while it is important, is not a common cause of diarrhea. There will also be no discussion of such conditions as the diarrheas associated with lymphopathia venereum or with chronic intussusception, or with new growths in which roentgenologic and sigmoidoscopic examinations should establish the diagnosis. A malignant origin must always be in the background of the physician's consciousness, especially in patients over forty years of age, when they occur most commonly. It is a sound rule to perform a digital and a sigmoidoscopic examination and to have a barium enema performed promptly in any patient in whom blood appears in the stools and cannot be explained by hemorrhoids; it is too frequent a sign of neoplastic disease to ignore. It is chiefly in the upper age groups that disaster may ensue by regarding diarrhea as a clinical entity and not as a symptom for which a cause must be sought.

CLINICAL PICTURE

The symptomatology of the three most important causative diseases, amebiasis, shigellosis and brucellosis, is remarkably similar. Common to all three are such symptoms as nervous irritability, easy fatigue, low grade fever, vague abdominal symptoms, arthritic manifestations, vague muscular pains and similar indefinite complaints. Both shigellosis and amebiasis may be characterized by regularly intermittent diarrhea, extending over a period of twenty-four to thirty-six hours and associated with malaise, frequently severe enough to require bed rest. If arthritic symptoms are part of the symptomatology, they are likely to be exacerbated during the diarrheal episodes, which have a notable seasonal incidence. In New Orleans exacerbations are most frequent between April 15th and June 15th and between August 15th and October 1st although why they appear at these times is impossible to explain.

Patients with disease of the colon are also likely to have undergone distinct personality changes. It might be added that their nervous status is in no wise improved by the fact that their complaints have frequently been diagnosed under a wide variety of terms and treated, without relief, by a variety of methods. One might be tempted, just as physicians previously consulted have been tempted, to discuss this variety of symptoms and syndromes under the general heading of neurosis, on the ground that they could not possibly arise from a single pathologic basis. The proof that they can, however, is two-fold: (1) the identification of the causative organisms in the stools in shigellosis and amebiasis and diagnosis by other measures in brucellosis and (2) the results secured in all three diseases in respect to symptoms by adequate treatment. This does not mean, however, that neurogenic diarrhea is not a clinical entity. The statement is fair that the larger the

number of such diagnoses in one's practice, the poorer are one's laboratory facilities. The diagnosis is practically always fallacious. A patient with chronic disease of the colon is likely to develop a chronic anxiety state against a background of chronic abdominal discomfort. But if the patient is thoroughly investigated it will usually be found that the history of colon disease is followed by the development of the chronic anxiety state and that the reverse sequence is infrequent.

Amebiasis. Amebiasis will be easier to comprehend if certain facts concerning it are borne in mind:

1. The parasite has a special predilection for two sites in the colon, the cecal and the rectosigmoidal areas, and the character of the symptomatology depends upon the location of the infectious process. Cecal infections are by far the most frequent. When they are present, amebiasis may assume the so-called syndromic form and simulate such intra-abdominal conditions as peptic ulcer or chronic appendicitis or cholecystitis. In infections in this area, constipation and not diarrhea is the outstanding symptom. When the infection is entirely or chiefly in the rectosigmoidal area, the disease takes a dysenteric or diarrheic form which may be either acute or chronic.

2. The passage of cysts is the rule in cecal amebiasis and the rather general belief that they are of no importance and that trophozoites are necessary for diagnosis (and for pathologic manifestations) is completely unjustified. In the opinion of the majority of American protozoologists, Endamoeba histolytica is a tissue parasite, not an inhabitant of the intestinal lumen. The "carrier" concept is therefore erroneous in this disease since tissue parasites are always pathologic and are never innocuous.

3. In neglected and badly managed subjects or in patients who develop lowered resistance for any reason, even subclinical amebiasis may assume a more serious form. Dysenteric symptoms may appear and may be followed by intestinal perforation and ameboma (amebic granuloma) as well as by hepatitis and liver abscess, the most common of the extraintestinal amebiases, or by involvement of other organs and systems.

Certain manifestations of amebiasis, all in children and all completely different from those in adults, have recently come to the writer's attention. This special symptomatology had not previously been observed, probably because it had not previously been investigated from the standpoint of possible amebiasis. Over the past eight months, in consultation with Dr. Maud Loeber, seventy-three patients with these special manifestations have been seen. The age range in the group was from six months to twelve years but the great majority of the children were under six years of age, a fact worth emphasizing because it has been the general belief that amebiasis does not occur in young children. Apparently, if one looks for it, it does.

The symptoms were bizarre and equivocal. The children, for the most part, were not really sick, they were just not well. While the symptoms varied widely, four characteristics of the condition were outstanding: (1) The complexion had a peculiar muddy, yellowish tinge which sometimes suggested fading suntan; (2) the liver was enlarged and tender; (3) personality changes were notable. Ordinarily normal, happy children, as well behaved as young children on the average are, became irritable, sullen and unmanageable. They had tantrums of temper. They struck attitudes of defiance. They seemed actually to take delight in baiting their parents and nurses; (4) the appetite was capricious and sometimes insatiable.

In addition to these quite constant manifestations, there were other more inconstant symptoms such as mild nausea and vomiting, low grade fever, headaches, increased fatigability, an increased susceptibility to colds, sometimes mild abdominal pain which woke the child from sleep or kept him from play, muscular pains in the arms, chest and legs and occasional, mild convulsive seizures of no recognizable pat-

tern. Slight constipation was frequent. Diarrhea was present in a few patients and mild dysentery in a few others. All laboratory findings were normal.

One might question the validity of these symptoms and especially their attribution to a single cause, except that in all seventy-three patients the diagnosis of amebiasis was made in the laboratory and was indubitable. Moreover, in every instance in which adequate therapy was instituted, there was prompt improvement in the symptoms.

The explanation of the occurrence of the symptoms (although for their pattern there is no explanation) was quite simple: In every case the parents, or others in close contact with the children, were found to have amebiasis which usually was not suspected and which unquestionably was passed on to them. When the diagnosis and then the explanation became apparent in the first few patients of the series, suspicion was aroused and the later patients offered no diagnostic difficulties. The infections in this group of patients were evidently light for identification of the parasites in the stools was on the whole unsatisfactory. The diagnosis could usually be made without much difficulty from aspirated material secured at sigmoidoscopic examination and sigmoidoscopy therefore was made part of the diagnostic routine. Undoubtedly, vague symptoms of the sort listed have gone unexplained in children in the past because the laboratory examinations which would elucidate them were not made. In the light of this experience, it would seem that pediatricians must add to their diagnostic regimen in cases of obscure symptoms in young children examination of the stools for ameba and sigmoidoscopy.

There was nothing particularly surprising in the experience just related. As one's experience with diseases of the colon increases, one becomes more and more impressed with two clinical facts: The first is that these diseases seem able to mimic not only the symptoms but the actual syndrome of many other diseases. The second is that the presence of amebiasis in particular seems to exaggerate and exacerbate symptoms of diseases with which it coexists although this may not be realized until the amebic disease is controlled and improvement in the companion disease becomes evident, without treatment directed toward it.

Amebiasis mimicks the combination of vague and inconstant symptoms already commented on which internists, for want of a better name, call neurosis or the anxiety state. It mimicks cholecystitis, appendicitis and peptic ulcer. It mimicks pelvic disease and it aggravates such gynecologic states as dysmenorrhea and the menopausal syndrome. It confuses the obstetrician when it appears in the third trimester of pregnancy when, for some reason, it is likely to become apparent. It aggravates sinusitis. Even the dermatologist is likely to find it entering the clinical picture. It is a disease, in short, which specialists in all branches would do well to bear in mind as a diagnostic possibility.

DIAGNOSTIC CONSIDERATIONS

The etiologic diagnosis of chronic diarrhea is a laboratory procedure. Nonetheless, the first step in the diagnostic routine should be to secure an accurate history. The investigation should begin with a careful inquiry into the patient's normal pattern of intestinal function. In diseases of the colon it is not the number of stools or their character but their difference from the usual norm which establishes the existence of the symptom.

As a rule, only suggestive, not conclusive information can be secured from the history but a history which brings to light nothing at all still has a negative value. It may throw light upon a number of important points. An inquiry into the bowel habits of the remainder of the family, for instance, should never be omitted. Even when habits of hygiene are good, it is not always possible to avoid the spread of an infectious disease and the information ob-

tained about other members of the family may be more revealing than the patient's own history. Occupation, dietary habits, the source of milk ingested, food dyscrasias, the relationship between symptoms and the physical and emotional state, places of residence, military service, these and other data may all provide clues to diagnosis which will save time, effort and expense.

Laboratory Diagnosis. The laboratory diagnosis of all of these diseases is difficult. It is missed in a very large number of patients because diagnostic attempts are not persisted in. Amebiasis has a cyclic pattern and if stool examinations are made during a period when cysts or trophozoites are absent from the stool, results will be poor. Sawitz's experience is typical; he found that in normally passed stools, the percentage of positive diagnoses progressively increased from 21 per cent when one stool was examined to 76 per cent when six stools were examined. Moreover, the examination of a single stool following saline purgation was as effective as the examination of three normally passed stools. In this condition culture technics give only inconstant results and the complement fixation method is impractical for general use because of the difficulty of obtaining potent and stable antigen.

Shigellosis is diagnosed by cultural methods, the cultures being prepared by a technic which provides uniformly desirable colonies for examination. The best results are obtained with less inhibitory media, such as McConkey's agar, eosin-methylene blue and Endo's agar. The percentage of positive cultures is higher from an enema specimen than from the stool or from material secured by aspiration at sigmoidoscopy. Occasionally, the organism is identified on the first culture. In the writer's experience, an average of five has been required and in some cases as many as twenty examinations have been made before positive results were secured. Diagnosis is likely to be simplified if the examinations are carried out during an exacerbation of symptoms but in any case the secret of success is repetition of the examination until the organism is isolated.

The diagnostic routine now employed for all patients who present themselves with a complaint of chronic diarrhea has evolved with experience, which has shown that the omission of any part of the study is likely to cause difficulties for patient and physician alike. The following investigations are carried out on successive visits:

1. Examination of a normally passed stool for protozoa and helminths; culture for Shigella of material secured from the rectal wall by the Hardy-Watt swab; tests for brucellosis.

2. Frei test for lymphopathia venereum; gastric analysis, to exclude achlorhydria; urinalysis; repetition of stool examination; repetition of culture for Shigella (Hardy-Watt swab).

3. Blood study, including mean corpuscular volume determination, to exclude sprue; repetition of stool examination; repetition of culture for Shigella (Hardy-Watt swab).

4. Sigmoidoscopy; examination of a stool passed after saline purgation for helminths and protozoa; culture of an enema specimen for Shigella.

The patient is prepared for sigmoidoscopy by a routine for which written instructions are supplied, as follows:

1. On retiring a dose of sodium phosphate sodium biphosphate (Fleet's phospho-soda) is taken. The cleansing effect is better than that of other saline purgatives and it seems to be ideal for encystation of Endanoeba histolytica; cysts are more practical for diagnosis than trophozoites. The medication is taken at night so that the patient is not aroused from sleep and so that the parasites during the night are washed from the lesions into the lumen of the bowel. If the purgative is taken in the morning, they have not had time for full maturation and are found as precysts, which do not lend themselves to differentiation.

2. A stool specimen is collected after 6 A.M. the following morning and is therefore fresh for examination.

3. Two enemas of physiologic salt solution in the amount of 1 quart each are taken in the morning; a 5 cc. specimen is collected from the final portion of the second enema. Culture of this specimen is particularly useful in the identification of Shigella.

4. Sigmoidoscopy is performed by the usual technic. Material is collected from demonstrable lesions if they are present and from random areas of mucosa if they are not. It is important that in amebiasis a special glass aspirator be used for the collection of the specimen; a cotton swab is not satisfactory because parasites are likely to be lost in the fibers.

A separate routine of testing is necessary for brucellosis. Cultural methods have been discontinued as not worth the trouble in chronic brucellosis although they may be valuable in the acute disease. It has been repeatedly shown, moreover, that the incidence of positive results is very low in abortus infections. The routine includes:

1. Agglutination tests, carried out by the Huddleston rapid slide macroscopic method with Lederle's antigen, which is quite as reliable and specific as slower technics: Different observers attach different degrees of importance to agglutination titers of various levels. Most observers regard titers of 1:80 as evidence of active Brucella infection while granting that they may occur in the presence of other infections and, of course, after skin tests for brucellosis or vaccine therapy with Brucella organisms. The writer formerly regarded all dilution titers of 1:160 or above as diagnostic of active brucellosis and titers below this level as suspicious. With increasing experience with the sensitivity test, however, an occasional patient with titers of 1:640 and more has proved not to have brucellosis. Several patients with titers of this level had negative sensitivity tests for active brucellosis; they were found to have amebiasis and were relieved of all symptoms with adequate treatment for that disease.

2. The opsonocytophagic index, which is based upon the ability of neutrophilic leukocytes to phagocytize Brucella organisms and which is an index of the degree of the patient's immunity rather than a true diagnostic test. Special laboratory facilities are required.

3. The intradermal test, by means of an intradermal injection of Castaneda's Melitensis, bovine, porcine vaccine. A positive test, which is presumed to indicate that the patient has, or has had brucellosis, depends upon the development of an erythema measuring more than 10 mm. at the end of twenty-four to forty-eight hours. The reaction is not regarded as negative until forty-eight hours have passed without the development of a clearcut erythema or of any erythema at all. A test which appears positive at the end of twenty-four hours but is negative at the end of forty-eight hours is interpreted as a negative reaction. In the writer's experience the intradermal test has proved a most satisfactory diagnostic method.

4. A sensitivity test, the principle of which is the injection at five-day-intervals of successively larger doses of the Castaneda vaccine until 2.0 cc. is reached. A single, clearcut reaction is regarded as diagnostic and therapy is substituted for further testing. If a clearcut reaction has not occurred before a dosage of 2.0 cc. is achieved, brucellosis can safely be eliminated as the cause of diarrhea. The reaction must be clearcut. A mild reaction is interpreted as negative for brucellosis.

Children are tested and treated with the Castaneda vaccine in the same dosages as are adults.

THERAPY

Initial Therapy. The first step in the management of a chronic diarrheal disease is, paradoxically, therapeutic, the administration of a multiple vitamin preparation of high potency. Diarrhea is a well known manifestation of such vitamin-deficiency diseases as sprue and pellagra and avitaminosis is a probable complication in a diar-

rhea of any origin which has lasted, to set an arbitrary limit for the establishment of chronicity, more than ten days. The hypothesis of a borderline avitaminosis is perfectly valid. When a person in this group develops diarrhea, it does not take long for the surplus of vitamins in the body, which is never very large, to be used up. The same process occurs in persons not in the borderline group whose diarrhea has been severe or has lasted over a considerable time. In neither type of patient can the vitamin needs of the body be met by the ordinary diet because, with a continuing intestinal disturbance, all food ingested passes rapidly through the intestinal tract and there can be no adequate absorption of vitamins. Even if the disease is self-limited, the deficiency continues to exist.

The patient with a continuous diarrhea, therefore, when he is first seen, is at once given a multiple-vitamin preparation of high potency. The results are frequently spectacular although they do not remove the responsibility from the physician for complete investigation. Incidentally, if the investigation proves negative and if there is no recurrence of the diarrhea, the results secured from therapy can justifiably be regarded as diagnostic.

Amebiasis. Every patient with amebiasis should be treated. This is not a disease which can be ignored with safety. The patient who harbors the infection is a source of infection for others. From his own standpoint he is either actually ill or is in a state of subclinical ill health from which serious complications may arise. It is open to question whether asymptomatic amebiasis very often exists. The patient classified in such a category frequently does not realize that he has had symptoms until he is relieved of them and enjoys the improvement in health and well being which follows.

In the order of effectiveness, the available amebicides are diodoquin, chiniofon, carbarsone and vioform although for some reason, which at the moment is not clear, diodoquin seems less effective than it formerly was. Emetine hydrochloride is of value in relieving the acute symptoms of either dysentery or diarrhea and it is the only drug effective in ameboma and in various extraintestinal amebiases. Its curative properties in intestinal amebiasis are probably less than 30 per cent.

Emetine, furthermore, is a protoplasmic poison and potentially toxic. Within a recent six-month-period, four patients were seen in consultation who, following its use, developed such severe symptoms that they had to be rehospitalized. The symptoms were numerous and bizarre and changed from day to day. They included cramps, headaches, dizziness and pains in various locations; they were associated with electrocardiographic changes and they were disturbingly slow in their disappearance. It may be that the dosage generally used at present (10 to 12 gr. in daily doses of 1 gr. each by the subcutaneous or intramuscular route; both are equally painful) is too high. As a matter of fact, very little is known about the optimum dosage; it is quite possible that effective results could be achieved with one-half or one-quarter of the dosage now being used.

If amebiasis is resistant to treatment and if after adequate investigation Shigella is not found in the occasional selected patient, combined treatment may be instituted (in the hospital), consisting of (1) emetine (1 gr. nightly for ten to twelve days) on the theory of eradicating the ameba via the blood stream; (2) penicillin (30,000 units intramuscularly every three hours for twelve days) to combat secondary infection via the blood stream and (3) neoprontosil (25 gr. four times daily for twelve days) to combat secondary infection via the intestinal tract. In addition, diodoquin is given three times daily in six-tablet doses while the patient is hospitalized, to eradicate ameba in the intestinal tract. The dosage is reduced by one-half after the twelve-day-period of hospitalization and is continued in that amount until the twentieth day. Neoprontosil is the best antidiarrheal drug presently available although its effect is probably on the associated pyogenic infec-

tion rather than on the amebic disease *per se*. Such combined treatment is extremely effective but it is also heroic and it should not be employed in the absence of special indications.

One point should be borne in mind in evaluating the therapy of amebiasis: Symptoms do not disappear until some time after parasites have disappeared from the stools. Post-treatment attacks, however, become successively milder until they eventually disappear altogether and therapy should not be reinstituted unless parasites again appear in the stools.

Shigellosis and Brucellosis. Sulfonamides and antibiotics are not effective in the chronic phases of shigellosis and brucellosis. Both diseases are treated with vaccines, administered subcutaneously in successively larger but cautiously increased doses at intervals of five to seven days, preferably the latter. The amount of the initial injection is gauged by the size of the skin reaction. Treatment of shigellosis is by an autogenous vaccine made up as soon as the organism is isolated. Treatment of brucellosis is by the M. B. P. vaccine (Melitensis, bovine, porcine) of Castaneda. In both diseases the patient is kept under careful observation, reactions to the previous injections are checked before succeeding injections, every patient is individualized and it is an invariable rule to employ a smaller dose whenever there is doubt as to the wisdom of a larger dose.

It is most important that these instructions be followed exactly. Dangerous reactions can ensue when they are disregarded and one fatality is known to have occurred under treatment in shigellosis which was charged up to the treatment but which should have been charged to the complete disregard of instructions.

An interesting observation during treatment in patients with brucellosis is the development of symptoms of which they had not previously complained, perhaps representing allergic reactions. If the explanation is valid, it supports the concept that the efficiency of Melitensis, bovine, porcine vaccine rests upon its desensitizing rather than upon its immunizing action. Reactions are likely to be severe if all foci of infection are not eliminated before treatment.

Vaccine therapy in shigellosis and brucellosis is supplemented by the Jones sugar-free diet which is strictly adhered to until improvement in symptoms occurs. Then articles from a list of permitted foods are added one at a time and in large quantities so that if an idiosyncrasy to them still exists, it will be demonstrated at once. If sensitivity no longer exists, these articles can be eaten in the usual quantities.

It is imperative that patients treated by vaccine therapy understand that this is not a rapid method of treatment. In shigellosis, results are not apparent for six months and in the usual patient treatment must be continued for a year or more although success in children is occasionally spectacular in much shorter periods of time. Patients with brucellosis are not likely to see any improvement before the tenth week at least. A minimum of six months of treatment is necessary, even if results are observed earlier, and treatment for a year or more is not unusual. Patients who have both amebiasis and brucellosis will not, as already pointed out, be benefited by amebicidal therapy until brucellosis is adequately treated. Prompt and permanent disappearance of Endamoeba histolytica from the stools can then be anticipated after a single course of amebicidal therapy.

In conclusion, two rather uncommon types of diarrhea might be briefly mentioned which undoubtedly exist but which are difficult to establish etiologically: The first is the so-called geriatric type, which occurs in persons over fifty-five years of age and in which neoplastic disease is not a factor. A possible explanation is the assumption by the intestine of new functions. The course of events is essentially as follows: Nephrosclerosis of various degrees is a rather common finding in most persons fifty years of age and over. Its existence is

reflected in dye excretion tests, the results of which are likely to be lower than normal. The diminished renal function, however, is not, as usual, reflected in the non-protein nitrogen level, which is substantially normal. The explanation may be that the colonic mucosa, which has the ability to extrude nitrogenous waste products, compensates by the assumption of this function for the decrease in kidney function. The irritation which is caused by the presence of the abnormal nitrogenous waste products in the bowel is expressed clinically as diarrhea. The theory is additionally supported by the fact that diarrhea is frequently a part of the syndrome of uremia. Patients in this group almost invariably improve when their protein intake is reduced.

The allergic variety of diarrhea undoubtedly exists but it should be regarded as established only after all other diagnoses have been excluded by adequate testing. It must be distinguished from the dietary difficulties associated with colonic diseases of other etiologies. Patients with chronic diarrhea, especially when amebiasis is the cause, very frequently cannot eat certain types of food but their difficulties are apparently not due to allergy for when the basic disease is cured by the proper therapy —therapy which would not affect an allergic condition—they can eat foods quite comfortably which formerly disagreed with them. Even more convincing is a personal experience with Jones' sugar-free diet, which is used routinely in shigellosis and brucellosis. Very few patients with diarrheas of other origins have not been relieved symptomatically by this diet. Yet it contains both wheat and eggs, which are common offenders in allergy and which cause no upsets when they are ingested. Patients with the allergic type of diarrhea are usually not seen in consultation. Food allergy is seldom the first manifestation of the allergic condition and it is a relatively simple matter to correct it in the course of treatment of the underlying disease.

SUMMARY

The chronic diarrheas have been discussed from the standpoint of considerations which have presented themselves as important in a practice limited to diseases of the colon. Amebiasis, shigellosis and brucellosis are the most common causes of chronic diarrhea. All three present vague symptoms, much the same for each disease, and all three are capable of mimicking other diseases. Diagnosis is a laboratory matter and may be difficult. The therapy involved has been outlined briefly.

DISCUSSION

Thomas T. Mackie (Winston-Salem, N. C.): I have found Dr. D'Antoni's paper extremely provocative. I am indeed sorry that the nature of his paper has prevented inclusion of evidence which I would certainly like to see in support of certain of his points. I am very much in agreement with many of the things that he has said but I am in serious disagreement with a number of others. It is impossible in the time allotted for discussion to do more than to touch upon the points of disagreement. I want to emphasize that this is merely the expression of differences in experience and differences in interpretation.

In the first place, with respect to the definition or rather the etiology of diarrhea and dysentery, I take exception to the statement that the etiologic agents are different. For example, the Shigella group and likewise the Endamoeba histolytica may produce, each of them, diarrhea and dysentery. I agree very strongly with Dr. D'Antoni's statement that Endamoeba histolytica is invariably a tissue parasite and that consequently it should be treated whether the individual has intestinal symptoms or not. By far the great majority of individuals who are carrying this infection fall into the category, unfortunately, of the individual who is classed as a neurotic because his symptoms are vague, indefinite and not characteristic of any well recognized clinical syndrome.

The type of picture which Dr. D'Antoni has described in children is important because it is a reflection not only of the picture in children but in many adults and one which I have been seeing in the last few months in many of our overseas personnel who are returning, finding

themselves incapable of resuming the normal intensity of civilian life because of an unrecognized and unthought of amebic infection. All patients then with amebiasis should be treated.

Now, we come again to one of the points with which I am in serious disagreement, the matter of the effectiveness of different drugs and the toxicology of these drugs. Diodoquin is useful and so are the other members of the hydroxyquinoline sulfonic acid group. These drugs, however, are not absorbed from the intestinal tract in such amounts to reach the amebae which are actually residents within the host's tissues. If one visualizes the pathologic condition as one sees it under the microscope, the way these protozoates are not only on the surface of the mucous membrane but are in the mucosa and deep in the muscularis mucosa, in the tissues of the submucosa, no drug which is not absorbed and distributed by the blood and lymph streams will be effective against organisms in that site. That statement applies to diodoquin, vioform and chiniofon as well as to the old German yatren.

The second group of drugs, of which there is only one member that should be used today, is carbarsone, an organic arsenical. That perhaps most nearly approaches the ideal amebicide because it is to some extent effective in both anatomic areas, that is to say the surface of the mucosa and within the tissues of the colon itself. But it is not completely effective and if one follows adequately—and by adequately I mean over a sufficient period of time—individuals resident in an area where they are in theory at least not subject to reinfection, one finds a relatively high recurrence rate.

Emetine, in my judgment, is certainly the most useful drug that we have because it is the only drug which is fully effective against the organisms which are in the tissues. Any of you who have treated amebic abscess of the liver know that. Its toxicology is very well defined. It is a protoplasmic poison; consequently, (and this is a general rule of therapy) any drug which is known to be toxic to host tissues should not be given in terms of so many grains per day or for a week, it should be given in ratio to the patient's body weight. That is fundamental. It has been thoroughly established with this drug that if the total daily dosage does not exceed 1 mg. per Kg. of body weight and if the total dosage over any course does not exceed 10 mg. per Kg. of body weight, one will not encounter toxic effects in the host that are a cause of any anxiety whatever.

The dosage that Dr. D'Antoni speaks of, 12 gr. in one course, in my judgment, is definitely higher than it should be. In his manuscript in the discussion of the complicating factors, and particularly the coincidental infection with the Shigella, the organisms of bacillary dysentery, he advocates the use of neoprontosil but in his paper of the morning he advises the use of vaccine. It has been the experience of the armed forces that the whole group of Shigella responds extraordinarily well to certain of the sulfonamide drugs. Perhaps the two or three most effective are sulfaguanidine, sulfadiazine and sulfathiazole. It seems paradoxical to me that he should recommend one of these drugs for infections complicating amebiasis and yet in his pure Shigella diarrheas advocate the use of autogenous vaccine.

Furthermore in my own experience, in long extended clinical trial of these cases using autogenous vaccines, I have found them not infrequently dangerous, as Dr. D. Antoni pointed out, but I likewise have been very unimpressed as regards their efficacy in eliminating the infection on the one hand or really relieving the patient's symptoms on the other.

This matter of brucellosis, as I have previously said, is one of the most provocative things in this paper. It is rather a curious situation it seems to me that with the importance which this disease has assumed in the United States since 1920 and the number of cases which have been reported that the syndrome of a chronic diarrhea due to that organism has not been recorded previously. That, of course, brings up the matter of diagnosis.

Dr. D'Antoni, I frankly think, rather unwisely has eliminated in his manuscript the one method by which a positive diagnosis can be made, in other words, the effort to recover the etiologic organism. The methods on which he depends are slide agglutination. The slide agglutination of itself is notoriously inaccurate. He depends upon the intracutaneous or cutaneous test using the killed organisms. That, if it is positive, merely proves an association and that association may not have been in its most active phase associated with a clinical response. It merely indicates that there has been a biologic relationship between a host and that particular organism.

He also includes the application of the

opsonocytophagic index, a procedure of considerable difficulty to carry out, the interpretation of which is still not without a certain amount of doubt and hazard.

The net result of it is that, in my judgment, there are only two methods by which a diagnosis of brucellosis can be made. The first, naturally, is the recovery of the organism; the second—and this is reasonably accurate—if one can show in the particular patient using the standard tube agglutination test that there is a rising titer of agglutins over a period of two or three weeks, then one may assume that one is dealing with an active and positive infection.

His treatment, again by vaccine, is disturbing. There are many treatments for brucellosis: Foshay's immune goat serum, all of the antibacterial drugs and antibiotics, the injection of cultured filtrates and, of course, the continued injection of autogenous vaccine over long periods of time. Again, there is a good deal of conflict in the experience of others as to the real efficacy of that type of treatment.

I want to say just a word about the giardiatic diarrhea. I would like to know if there is any evidence for it. I personally have never seen a situation in retrospect that I could possibly explain on such a hypothesis. I question the wisdom of submitting such sweeping hypotheses with no evidence in support.

May I conclude then, by complimenting Dr. D'Antoni on a very interesting paper and repeat again that what I have said is not meant in any sense as unkind criticism; it is simply a frank expression of my own interpretations of these problems.

Mary M. Spears (Philadelphia, Pa.): I am very much interested in Dr. D'Antoni's paper on chronic diarrheas, particularly as it relates to their etiology.

The incidence of the types described by the author is small in Philadelphia compared to that in New Orleans. The patients I see with chronic diarrhea fall into a miscellaneous group etiologically, with a low percentage due to infection. I should like to confine my remarks to the cases with which I am familiar.

I agree with Dr. D'Antoni that chronic diarrhea is not a clinical entity but is an expression of an underlying cause which is not necessarily in the colon. Since the etiology is difficult to determine, the patient should be considered as a whole and not from the condition of the colon alone. Also emphasis should not be placed entirely on the laboratory for diagnosis.

Obviously a careful history of symptoms, including eating habits, personal hygiene, type of employment and a thorough physical examination are important. While the social status is important, I have found that diarrheas do not occur exclusively among those who eat too little and therefore suffer with malnutrition, but perhaps more frequently among those who eat too well. A diary of food and symptoms is often very illuminating and will throw convincing light on the cause of many cases.

Allergy as a causative factor in diarrhea cannot be dismissed. From my observations allergy is quite near the top of the list in the etiology of this condition. Some of the offending foods that have been exposed are wheat, citrous fruits, dairy products, sugar, carrots and some drugs. In this connection I think it is important to know what medication the patient has been taking. We have found the diarrhea in a given case to be cured by the withdrawal of sedatives and vitamins to which the patient was allergic.

We should know something about the emotional background, incompatibilities and conflicts in the homes and the places of employment of our patients. In the patients in whom the emotional basis is the factor the condition is secondary to the anxiety state and not the anxiety state secondary to infection. It has been demonstrated many times that when the environment has been changed and the situation relieved the diarrhea has promptly cleared up.

A physical examination has revealed an undiagnosed hyperthyroidism which was the cause of the diarrhea for which the patient sought relief: a cure took place when the underlying cause was treated. The cholecystectomized patient is often the victim of a diarrhea which is resistant to treatment.

A gastrointestinal investigation should include biliary drainage, gastric analysis, feces examination and rectosigmoidoscopy. The first will sometimes expose giardia which in some of the cases we have seen have been the etiology; achylia has been the cause in some patients and carbohydrate fermentation the basis in a large percentage. We are all familiar with the tragedies of patients who have been treated and not examined and in whom an inoperable carcinoma is found. In our patients

who have all had complete studies the number due to bacterial infection or parasitic infection is very small. X-ray examinations were done as indicated.

I cannot recall a patient with diarrhea due to avitaminosis, but I have treated patients with it caused by a vitamin. I think we should be careful and alert in our medication.

I appreciate the privilege of participating in the discussion of this interesting paper and this important medical problem.

Martin S. Kleckner (Allentown, Pa.): It has afforded me great pleasure to hear Dr. Antoni's paper on chronic diarrheas. As a man who has had tremendous experience in the field of parasitology and pathology, he has brought to us a review of some of the important phases produced by this symptom-complex. I do not agree in full entirely with everything that Drs. D'Antoni or Mackie have said so that I will pursue a middle of the road attitude. I have found this very interesting, and at times, distressing symptom to exist in 15 per cent of my patients in protologic practice.

Due to airplane travel and the return of many of the servicemen following peace in the last world war, many of the tropical diseases which had not been as prevalent heretofore have either been greatly increased or their presence has been brought more strongly to our attention.

I wish to enumerate several points concerning which you as proctologists treating diseases of the anus, rectum and colon must always be cognizant: Careful history taking is very important as well as complete physical and proctologic examination which includes sigmoidoscopy. We are dependent in great measure on the pathologic and roentgenologic laboratories. Those of us who are fortunate enough to have trained personnel in our laboratories that understand parasitology and those who believe in the diagnostic value of the double contrast barium air enema are indeed most fortunate.

Examination in the pathologic laboratory of warm stools from clean containers produces the best results if analyzed within the first forty-five minutes. I do not believe the taking of smears for culture and scrapings via the sigmoidoscope has been mentioned.

Diarrhea is due to a number of causes. Some of them may border on the acute but we must bear them in mind. They are as follows:

1. Ingestion of improper food and drink. Food poisoning and prolonged overindulgence of poor grade liquors should be mentioned.

2. Deficiency diets when avitaminosis plays an important part. Pellagra is a good example.

3. Diarrheas due to invasion and infections by certain organisms of bacterial and protozoal origin. My experience is very limited regarding shigellosis and brucellosis but the amoebic and other forms of bacillary dysentery have been seen. These must be remembered in diagnosing the diarrheas. Do not forget that the "carriers" of these conditions may need more personal care than many others. Certainly our health examinations in regard to handlers of food and drink should receive more attention than ever. Stool cultures and smears are often disregarded in this modern era when health and longevity are emphasized. Under this category must be mentioned chronic ulcerative colitis many times characterized by eight to fifteen daily bowel movements.

4. Giardiasis and the malarias are mentioned because they may produce severe chronic diarrheas.

5. Anatomic derangements or lesions in the colon such as diverticulitis, the presence of polyps and malignancy may all cause chronic diarrhea. One thing we often forget is the presence of the most common foreign body in the rectum, fecal impaction. Some of these very large impactions in patients may produce a diarrhea, or a tenesmus simulating diarrhea for weeks and may require the injection of peroxide of hydrogen, oil enemas, etc., to break it up. Sometimes a patient must be put under an anesthetic in order to evacuate the impacted feces.

6. There are various nervous influences that may produce a diarrhea and we are prone to call it (erroneously) mucous or functional colitis.

7. Long administration or overdosage of certain medications such as arsenic and mercury and the much abused mineral oil. The active use of radium and deep x-ray therapy may cause what is known as a factitial proctitis with diarrhea as a common symptom.

8. Parasitic infestation listing the helminths, the cestodes and the nematodes as primary causes is common but so little understood. I regret that time does not permit further elaboration of this condition.

Let us insist upon complete investigative methods that all chronic diarrheas may be more carefully diagnosed and treated.

Joseph S. D'Antoni (closing): I think it is most unfortunate that I have been negligent over the years in publishing a lot of the work that we have been trying to do and tried to get to the bottom of. Consequently, I think that some of Dr. Mackie's statements could be blamed on this fact; namely, that it is introduced in very short sentences and very little or no background for our having come to those decisions has been made in the paper. I am not blaming him for not reading the literature. I am blaming myself for not having published some of this work and given more definite proof, as he asked for.

First of all, regarding diarrhea and dysentery and my statement saying that it should be differentiated because the etiologies are different, I am speaking at that point of the group of etiologies. When you have dysentery, your patient is automatically transferred into a particular group and when you have chronic diarrhea, he is thrown into another group; when you have acute diarrhea, he is thrown into a third group. I did not say that the etiologies in all of these groups were different. I have taught for years and believe I am correct in the statement that in the southern United States, 90 per cent of all dysenteries in the acute and chronic form are due either to Levick or Shigella. In the northern United States, that statement would have to include your nonspecific ulcerative colitis which, as I have said, we do not see. In certain parts of the area where flukes are present, namely, the Schistosomum mansoni infections and japonicum infections, then if you have dysentery, you must include it as one of the etiologic agents. But it has always surprised me intensely to have patients with dysentery referred and a diagnosis made of some etiology that cannot produce dysentery. If the man who referred that patient had known that many of these etiologies do differ, that mistake would never have been made.

As to the effectiveness of drugs, of course, I have been one of the big boosters in this country of diodoquin. The British have used it recently. So in contradiction to Dr. Mackie's statement that iodine preparations will not cure, for the first time in the history of our knowledge of amebiasis the British are curing 75 per cent of their amebic patients who live in India, renowned for the most resistant strains in the world. If they add penicillin to it—and penicillin, as I have said in the paper, just as neoprotosil, is used to combat secondary infection—that cure rate has been made much higher.

I frankly doubt and I have one very capable backer in the person of Colonel Craig who has made the very definite statement that he doubts that 20 per cent of the patients with amebiasis are cured with emetine. I more or less have to agree with him on that basis aside from its toxic effect. I see Dr. Sodeman in the audience who can vouch for one lady who on the second grain of emetine (a woman of about 128 pounds) developed paroxysmal tachycardia and electrocardiographic changes. We have seen a number of patients of that type and we expect to see more if we are going to continue using this drug.

I made the statement very definitely in my paper that I believed neoprontosil was the best antidiarrheal drug, meaning that it was given to combat a symptom and not as a curative agent.

Secondly, we have always used autogenous vaccine and have always believed it to be the most efficacious.

On the question of brucellosis, Dr. Mackie, we have—and I reported at the Congress on Brucellosis in Mexico last November—twenty-six patients with intestinal brucellosis who had been observed for a period of six months to three and one-half years under vaccine treatment. These patients, I believe, have gone sufficiently long to say most probably we are correct. We have to wait ten or fifteen years probably to say that unequivocally but on the basis of what I have seen of diarrheas, on the basis of what you can learn in the literature regarding brucellosis, if you read about the pathologic conditions you will find that every organ in the body has been found to harbor this bug. I would like to be able to say that we can culture 90 per cent of the organisms present in all those with brucellosis. On the other hand, I doubt very seriously if we can culture the causative organism in more than 2 or 3 per cent.

I think in this country that 10 per cent of the chronic abortus infections can be cultured from the blood stream and that is by some very capable workers such as Huddleson, Forshay, Harris, Alice Evans and so forth. If we are going to rely on the culturing of brucella abor-

tus from the blood stream, then we are going to have more neurogenic diarrheas, as I have already stated, and stated why I thought they occurred so frequently.

Now, the vaccine that Castaneda has proposed is an endotoxin. I did not discuss it in the paper because it is in the American literature, primarily in the American Journal of Tropical Medicine.

As to the giardiatric diarrhea, I could defend myself by telling you a personal experience that occurred between my junior and senior year as a student. I worked in the pathology department at Charity Hospital and during the three months' period we had, I believe, 225 postmortems that were usually done within thirty to forty-five minutes after the patients had died. In those days the independent service in the hospital did all the postmortem examinations and we made it a rule to get through with these examinations as soon as possible so as to keep in the good graces of the undertakers. In seeing the bowel of uremic patients and in seeing the bowel of people over fifty years of age who had diarrhea, we frequently saw a yellowish, brownish-yellow discoloration in the submucosa and no textbook on pathology would tell us the pathologic condition that was in process. For some very peculiar reason which I am at a loss to explain, and I have asked many pathologists for an explanation, this brownish, peculiar submucosal discoloration disappears about one hour to an hour and one-half after the patient is dead, so that you must obtain the bodies "hot," in other words, in order to see this.

The fact that the colonic mucosa is capable of extruding nitrogenous waste products I think is an accepted fact among pathologists. I have limited it here to the colon. However, most pathologists believe and have shown, and it is a common fact among them, that nonprotein nitrogen is extruded through the mucosa of the gastrointestinal tract.

There is one thing I would like to say. In the paper I have tried to include as many things as possible simply to develop in the physician an interest in the chronic diarrheas instead of this procedure of throwing his hands up and saying, "Neurosis!" My secretary will tell you that I have the craziest people or more crazy people than any three psychiatrists in New Orleans, but it is remarkable how, after you rid them of their colon disease, mentally well these individuals become.

My remarks are not limited to patients from New Orleans. I have had the fortunate experience of seeing many patients from the tropics and from all over the United States. In Philadelphia, as in every other part of the United States, amebiasis is present. If you have a diarrhea—and I do not care what part of the United States or the world it is—the first thing you think of is amebiasis. I think if you continue looking for it and can get your doctors and the pathologists interested in it, you will find a much higher incidence than is presently reported.

I made the statement and thought I had made myself clear regarding the allergists. My experience has been this, that most patients are referred by other physicians and since they are referred and since so many of them have been to three, four, five, eight and ten doctors, the allergic diarrheas have already been picked up before we ever see them. I think there is no question but that it does very frequently occur. In my experience, and for the reasons which I have just enumerated, I do not see them very often.

As to the question of physical examination in these patients, I am not proud of the fact but because of the great tediousness of this work I have been unable to do complete physical examinations on these individuals, but I have always made it a practice to see if the physician recommending or referring the patient has not put that patient through the mill; most of them, incidentally, have been in hospitals five or six times; if the physician referring the patient has not had that patient in the hospital and given him a good work-up, he is always referred to a capable internist. We never begin looking for the cause of diarrhea until that internist will tell us, "Well, I think it is colon infection," not coming from such systemic disease as endocrine glands and so on and so forth.

Several years ago a urologist sent me a patient who had arthritis of the right hip. In subjects with shigellosis, we have seen a few patients with colon infection that had marked hypertrophic arthritis and because of the results that we obtained with this patient, one in particular and two others that were from this one urologist, he became very conscious of

the fact that if patients have colon disease, they may have a shigellosis and in turn that may be what is aggravating their arthritic manifestation. He sent me such a patient. The patient had not had a physical examination in several years. Therefore, I sent this patient to an internist. The internist examined the hip and found that there was marked limitation of motion. She had been having this trouble for about three months. He ordered an x-ray first and found that the femur was fractured and that was the arthritis that was being treated.

Consequently, I have a firm belief that all patients should be seen by an internist and the most capable one that you can find, if you do not do them yourself.

As for the giardial diarrheas, several years ago we were quite interested in this and checked back on our figures. In 5,000 patients with giardiasis that we had seen, over approximately a nine-year-period, there had been only three that had diarrhea from Giardia. Consequently, we always wait until we have ruled out every other etiology and if we can find no other etiology, then we say it may be the Giardia and treat the patient accordingly.

As to Dr. Kleckner, I would like to bring out one point which I did not mention in my paper. That is the complaint of the returning serviceman. His complaint is usually severe abdominal cramps and a tendency toward constipation rather than diarrhea. I would guess that in the probable seventy-five or one hundred returning servicemen I have seen, about 75 or 80 per cent of them have had this one complaint of abdominal pain which is not at all infrequent but it is certainly not that common in all patients with amebiasis.

Regarding some of the topics or some of the diseases that Dr. Kleckner mentioned, we have made it a habit to place those in the acute diarrheas such as food poisoning, the poisoning of cadmium and other chemicals, such as arsenic and fluoride, and so forth. They, I believe, come under the heading of the acute diarrheas rather than the chronic diarrheas.

Likewise, I believe that fecal impaction, the patients I have usually seen, rarely run for a month. Consequently, they, again, are transferred into the group of acute diarrheas.

Finally, I would like to comment on the helminth infections: Needless to say, schistosomiasis is notoriously a producer of diarrhea and dysentery. The Strongyloides infections, which are fairly common in this country, also notoriously produce diarrhea but of the helminths, we can say this in general, any helminth is capable of producing diarrhea. Consequently, if you can find no other cause, regardless of how light your infection is, if you find, for example, only one or two eggs of hookworm on a zinc sulfate centrifugal flotation specimen, treat the patient for those hookworms, because that may be producing diarrhea.

In summary, let me again thank the three doctors who discussed this paper.

EVALUATION OF THE ROENTGENOLOGIC DIAGNOSIS OF LESIONS OF THE RECTUM AND SIGMOID

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Toledo, Ohio

LIKE many familiar phrases, that which defines the specialist as one who grows to know more and more about less and less is not as trite as it sounds and certainly contains some elements of truth and common sense. Those of us in the more limited specialties must be constantly alert to maintain a sound sense of balance.

The purpose of this report is to emphasize the need for co-operation between the roentgenologist and proctologist in that small group of cases in which all available information is necessary in arriving at an accurate diagnosis. We can only deplore the prevailing tendency on the part of the patient as well as his attending physician to accept the negative x-ray findings as infallible.

It is not within the scope of this paper to dwell in detail upon any of the technical phases of x-ray examination of the colon. However, emphasis should be placed upon the diagnostic superiority of the air-contrast technic over the routine barium enema, particularly when the latter has failed to show any pathologic condition in the presence of definite symptoms. The air-contrast examination requires additional time and effort on the part of the roentgenologist and is often an extremely uncomfortable experience for the patient. Nevertheless, the judicious employment of this technic will provide an accurate diagnosis resulting from the persistence, patience and skill of the conscientious roentgenologist. The latter is always ready to point out to the family physician the diagnostic inadequacies of the x-ray in the rectum and rectosigmoidal area and frequently suggests further examination by direct vision through the sigmoidoscope.

In order to illustrate the advantages of close co-operation between the roentgenologist and proctologist, representative cases have been selected from that group in which every possible diagnostic aid has been necessary to attain the final diagnosis. Although the space restriction permits the use of only one plate for each case, several films were taken of every patient chosen for presentation here.

Congenital Malformations. The x-ray can frequently furnish valuable preoperative information concerning various congenital malformations of the anus and rectum. When called to see a newborn baby with an imperforate anus and no demonstrable outlet, the surgeon is confronted with a surgical emergency that calls for all the diagnostic aids at his command. It is particularly in a deformity of this type that x-ray is useful in demonstrating the location of the blind sac. Knowing this, it can readily be determined whether the sac can be reached through a perineal incision or whether it is so high that such an approach would be useless.

The maneuver suggested by Wangensteen and Rice[1] for determining the distance of the blind sac from the perineum in a newborn infant with an imperforate anus and no demonstrable outlet is a very useful procedure. The infant is held by his feet long enough to allow any gas that might be present to rise and accumulate at the blind end. The relation of the air bubble to an opaque object fastened to the perineum determines the level of the blind sac. The writer has found this technic useful on three occasions.

CASE REPORTS

CASE 1. In this first case a congenital atresia of a segment of the colon was suspected.

The infant was seen about eight hours after delivery and during that period no meconium had been passed and the abdomen had become distended. A normal anal outlet was present and a catheter could be inserted for a short distance. A plate (Fig. 1) was taken to see if any relationship could be shown between the level of the catheter tip and an air bubble at the obstructed area. This could not be demonstrated and the film served only to confirm the impression that any obstruction present was well within the abdominal cavity.

Case II. A newborn infant had an imperforate anus with a perineal sinus so small that only a small probe could be inserted. (Fig. 2.) A dilute solution of a radiopaque oil was injected into the opening to demonstrate the low lying sac shown here. Knowing the position of the blind gut, it was possible to reconstruct an anus at the normal site at once rather than to temporize by enlarging the perineal opening.

Case III. A nine year old girl was seen for the first time with symptoms of a low grade obstruction. Her only terminal outlet was a perineal sinus too small to admit an examining finger or an instrument. After the obstruction had been relieved by medical measures the x-ray demonstrated the tremendously dilated left colon pictured in Figure 3. It was evident that the dilated large bowel was caused by chronic obstruction at the outlet and was ap-

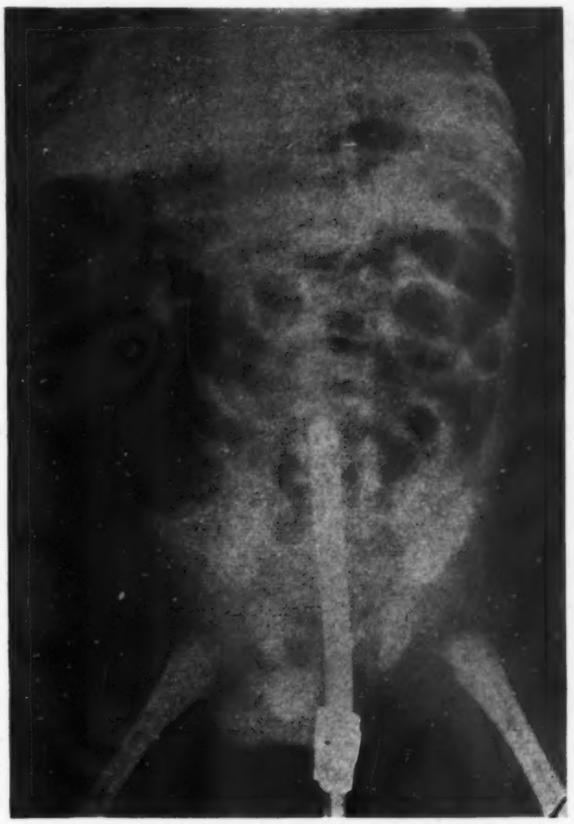


Fig. 1. Case 1. No air bubble could be demonstrated to show the site of the obstruction but the position of the catheter confirmed the impression that the point of obstruction was well within the abdominal cavity.

Fig. 2. Case II. Opaque media injected into the perineal sinus of a newborn with an imperforate anus revealed a low sac that could be approached through the perineum.

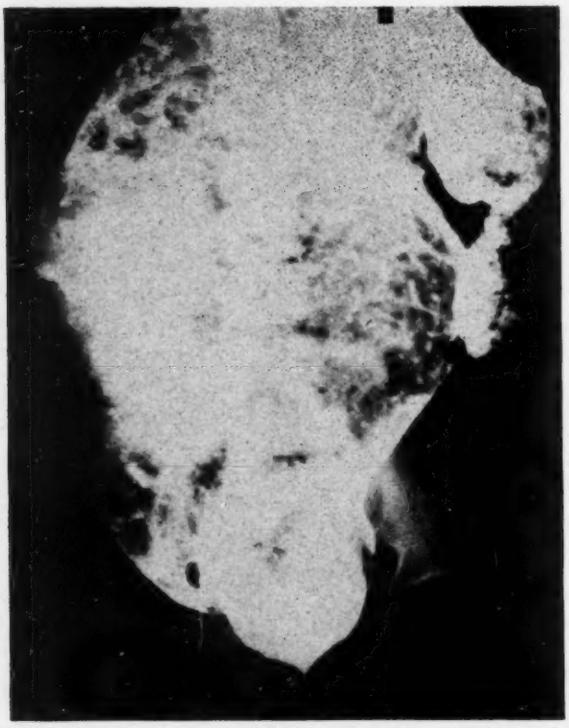


Fig. 3. Case III. The dilated left colon in a nine year old girl was readily demonstrated by x-ray although the only bowel opening was a perineal sinus too small to admit an examining instrument.

parently not a true case of congenital megacolon.

Lesions beyond Reach of Sigmoidoscope. The value of the roentgenographic findings in those patients where the lesion lies beyond the reach of the sigmoidoscope is obvious. However, two cases are presented to illustrate the dependence upon the x-ray in accurately diagnosing lesions above the lower sigmoid.

Case IV. The plate shown in Figure 4 was taken as a routine procedure following the sigmoidoscopic discovery of the pedunculated adenoma that is demonstrated on the film just above the rectosigmoid. The presence of a second polyp beyond the reach of the instrument demonstrated the necessity for an abdominal approach although the lower lesion could have been removed through the operating sigmoidoscope. An interesting side light in this case was the fact that the roentgenologist, by maneuvering the second polyp under the fluoroscope, was able to estimate the length of the pedicle to within a centimeter of its actual size as later found at operation.

4

5

Fig. 4. Case IV. The arrow points to two adenomas of the sigmoid, only one of which could be reached through the sigmoidoscope.

Fig. 5. Case V. This film gives x-ray confirmation of a tumor of the descending colon tentatively diagnosed on sigmoidoscopic examination by the presence of blood-tinged mucus in the lower sigmoid.

Fig. 6. Case VI. Roentgenographic evidence of a chronic supralevator cavity complicating an old ischiorectal abscess. The cavity above the levator had not been detected at two previous operations to obliterate the ischiorectal sinus.

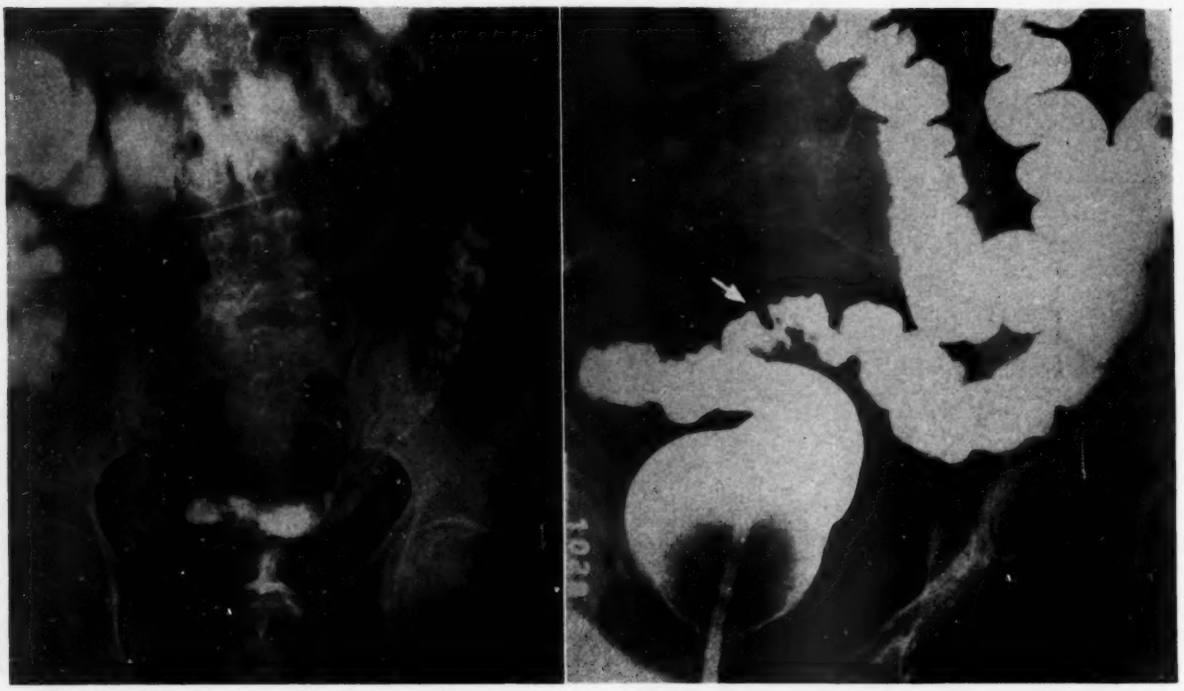
Fig. 7. Case VII. Although this nine year old boy had had three unsuccessful previous operations for a fistula-in-ano, the x-ray showed that the persistent tract was a fairly simple one.

Case V. In this case the x-ray confirmed the tentative diagnosis of a tumor of the descending colon and demonstrated its approximate location prior to operation. (Fig. 5.) On earlier sigmoidoscope examination, only blood-tinged mucous was found to suggest the presence of a lesion beyond reach of the instrument.

Fig. 8. Case VIII. The presence of the opaque material anterior to the coccyx proved the existence of an unhealed cavity although the patient had been discharged as healed the day previously.

Fistulas. Although the ordinary diagnostic measures are usually adequate to provide all the information necessary in the management of anorectal fistulas, the surgeon is occasionally confronted with a patient presenting an unusual problem. Three such cases have been selected to illustrate the additional information to be gained from an x-ray examination of the tract following the injection of a radiopaque oil.

Case VI. About six months before this film was made (Fig. 6) a large ischiorectal abscess had been drained in the usual manner. A subse-



9 10

FIG. 9. Case IX. This was one of several films which failed to show the presence of a carcinoma just above the rectosigmoid; the diagnosis was made on sigmoidoscopic examination.

FIG. 10. Case X. The lesion at which the arrow points was diagnosed by x-ray and fluoroscopic examination as a carcinoma of the sigmoid. The sigmoidoscopic appearance, augmented by biopsy, proved that the defect was caused by fibrosis secondary to a chronic diverticulitis.

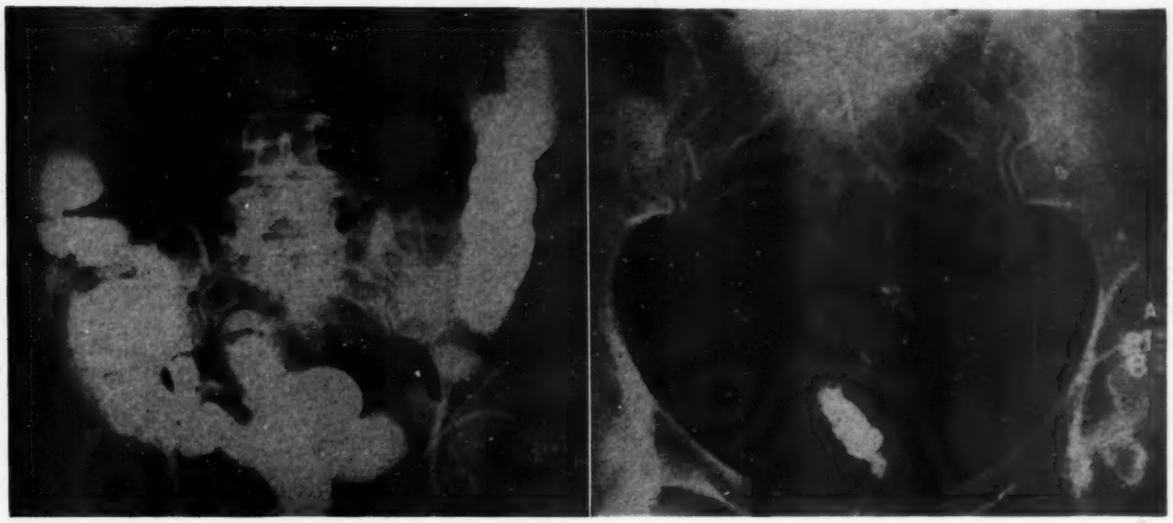
FIG. 11. Case XI. What appeared on x-ray to be a large polyp of the lower sigmoid proved on sigmoidoscopic examination to be a knuckle of sigmoid intussuscepting through the rectosigmoid into the rectal ampulla.

quent fistulectomy with obliteration of the internal opening was carried out about five weeks later. Healing progressed to a point where a small sinus persisted in the scar over the ischiorectal fossa. Probing afforded no information as to the reason for incomplete healing. However, the injection of an opaque oil demonstrated a large supralevator cavity which had not been discovered at the two previous operations.

CASE VII. Prior to the first examination, a seven year old boy had had three unsuccessful operations for a fistula which was first noticed before he was a year old. Figure 7 demonstrates a fairly simple tract which enabled the surgeon to begin the operation with a feeling of confidence he might not have otherwise had.

CASE VIII. The patient was seen the day after he had been discharged as healed by a surgeon who had six weeks previously performed a fistulectomy. A defect in the scar was found and the injection of an opaque oil demonstrated the cavity shown in Figure 8. Because of the possibility that the case might develop an important legal aspect, the x-ray was valuable as additional evidence to substantiate the diagnosis.

Incorrect X-ray Diagnosis. Up to this point the cases presented have illustrated conditions in which the proctologic diag-



A B

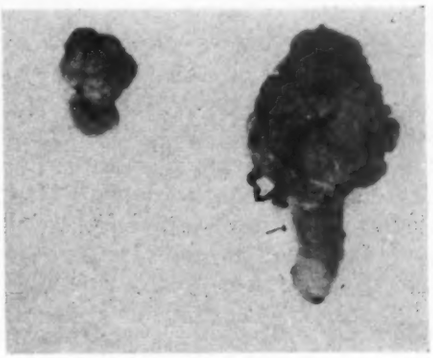
FIG. 12. Case XII. A, this film of a routine barium enema failed to show any disease in the sigmoid area; B, an air contrast study on the same patient revealing one large polyp in the sigmoid.

nosis has been supplemented and aided by the x-ray findings. The following group is composed of instances in which the x-ray diagnosis was inaccurate and the correct diagnosis was reached only after a sigmoidoscopic examination had been made.

CASE IX. This case is presented to emphasize the inadequacy of the x-ray in accurately diagnosing lesions in the area just above the rectosigmoid. In this instance no lesion was discovered on x-ray but the symptoms were such that proctologic consultation was requested *at the insistence of the roentgenologist.* Sigmoidoscopic examination demonstrated a carcinoma in the area above the rectosigmoid that was difficult to visualize on x-ray examination. (Fig. 9.)

CASE X. A roentgenologic diagnosis of carcinoma of the lower sigmoid was made after careful and repeated x-ray examinations. However, when a sigmoidoscope of small caliber was passed into the lumen of the stenosed area, pus was seen escaping into the bowel from a small diverticula opening; a biopsy specimen was also taken from the wall. The defect, diagnosed as a carcinoma on x-ray examination (Fig. 10) was shown to be caused by the fibrous thickening of the wall secondary to a chronic diverticulitis.

CASE XI. This case was a beautiful air-contrast demonstration of a polyp prolapsing into the rectal ampulla through the rectosigmoid. The child was first seen by a pediatrician who



C

FIG. 12. Case XII. C, the larger polyp pictured in this photograph is the one shown in the air contrast film. (Fig. 12B.) The second and smaller polyp was not demonstrated by x-ray but was found at operation just 2 cm. above the larger one.

replaced what he thought was a polyp that was protruding through the anal orifice. The x-ray film (Fig. 11) confirmed the diagnosis and it was decided that such a low-lying tumor could best be removed through the operating sigmoidoscope. The age of the child was such that the passage of the scope without anesthesia was inadvisable. Preparations were made to remove the tumor under anesthesia and the scope was passed to a distance of 20 cm. from the anal margin, a point well above the site of the polyp as shown on x-ray examination. However, no tumor was seen although its presence was again confirmed by another x-ray. At this point, a general surgeon who was called in consultation felt that the roentgenographic find-

ings justified an exploratory laporotomy. At the insistence of the child's father who was a physician, another attempt was made to locate this apparently low-lying polyp. When it was not found, the anesthetist was asked to lighten the anesthetic so that the child would strain and push down any tumor that might lie above the reach of the scope. No polyp was seen but as the instrument was withdrawn a knuckle of sigmoid intussuscepted through the rectosigmoid to simulate the "polyp" so clearly demonstrated in Figure 11.

Case XII. The three photographs in Figure 12 belong in a series that again emphasizes the fallacy of complete acceptance of the roentgenologic findings. Figure 12A is that of a routine barium enema ordered because of occasional, slight bleeding with defecation; nothing was found to explain the symptoms. After the examination was repeated using the contrast technic the presence of the large tumor seen in the lower sigmoid was clearly demonstrated in Figure 12B. The larger of the two polyps shown in the photograph (Fig. 12C) was the one seen in Figure 12B after it had been resected through the sigmoidoscope. Following its removal, the smaller one was discovered less than 2 cm. above it. It was apparent that the larger tumor had completely obscured the smaller one.

Psychic Factors. This presentation would not be complete without mentioning that large group of patients in whom an x-ray examination of the colon is necessary to convice them that no disease exists. Roentgenographic findings in this group are usually negative. In these times when the subject of cancer is so well publicized all physicians are familiar with the apprehensive individual, usually afflicted with a spastic colon, who will accept the fact that no tumor exists only after he has undergone a colon x-ray. Some individuals who have had an abdominoperineal resection for carcinoma of the terminal bowel are sometimes obsessed with the idea that they have developed another cancer. On a few occasions it has been necessary to do a colonic filling through the colostomy stoma in order to convince these unfortunate patients that no other lesion exists. A thorough physical examination, including the passage of the sigmoidoscope, fails to impress them as does the huge machine that crackles and sputters in the darkness of the fluoroscopic room.

CONCLUSION

This has not been the type of report from which one can draw any well defined conclusions. Each physician must determine for himself from the history and physical findings in the individual case whether or not an x-ray examination will be of benefit to his patient. It is a waste of time and money to order colonic fillings routinely just as it is criminal neglect to omit that examination if it can be useful in arriving at an accurate diagnosis. By working in close cooperation in the type of cases mentioned here, the proctologist and roentgenologist can arrive at a more accurate diagnosis than either could have reached alone.

Credit is given to Drs. M. E. Goodrich, W. S. Peck, C. E. Hufford and F. C. Curtzweiler, of Toledo, for the use of their films for reproduction. Close cooperation with these roentgenologists has been stimulating to the author and has resulted in a more accurate diagnosis in the type of cases presented.

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DISCUSSION

Jerome Wagner (New York, N. Y.): The privilege of commenting upon the presentation of Dr. Green is an opportunity to add emphasis to our obligations and duties as proctologists and to disseminate to the medical profession the knowledge we have acquired in our special field. In a paper read before the Gastroenterologic-Proctologic Section of the American Medical Association in July, 1946, entitled "Roentgenologic Contribution to the Diagnosis of Functional Intestinal Disorders," Dr. Harry M. Weber of the Mayo Clinic said, "If abnormality is suspected to exist in the rectum or sigmoid colon, the proctosigmoidoscopic examination is done first. This examination has obvious and insuperable advantages over even

the most competently performed roentgenologic examination in the investigation of these portions of the large intestine."

The roentgenologists and the proctologists have learned that a barium enema, in the quest for rectal and lower sigmoidal pathologic symptoms, is the improper procedure, before a digital or instrumental examination has been performed. It is still common practice to request a gastrointestinal series and barium enema for a patient before proctologic study. Suspicion may then arise that warrants the advisability of a proctologic examination. The proctologist's first findings, then, are a bowel wall coated with barium, or, as frequently happens, particularly in elderly persons, a fecal impaction.

As Dr. Green has demonstrated, there is a very definite place in proctologic practice for the x-ray as a diagnostic aid, particularly air-contrast studies but, like all laboratory aids, it should be utilized as such. X-ray studies above the mid-sigmoid are invaluable, for example, as shown in recent advances made in cecal studies of amebiasis; but caudad to the mid-sigmoid, not only are they of limited value but they may often be misleading.

In view of this, proctologists must stress the words of Dr. Weber which I have quoted. They must stress the simplicity and extent of a proper digital examination, bearing in mind the teachings of that master surgeon-anatomist, John B. Deever, who stated, "The relative positions held by the terminal part of the sigmoid flexure and the first portion of the rectum should be borne in mind, for, with a finger in the rectum quite a small growth in the terminal part of the sigmoid flexure or in the terminal part of the mesosigmoid can be detected, not that the point of the finger is in immediate contact with the growth, but that the tumor can be felt through the intervening wall of the first portion of the rectum." This is the area that roentgenologically is most confusing and is often referred to as the blind area.

Dr. Green's demonstration has been a lucid exposition of his subject.

Andrew Taylor (Hartford, Conn.): The subject matter of Dr. Green's paper affords very little, if any, opportunity for discussion but it does provoke a few thoughts which I would like to pass on as an appendix to his paper, with his permission. At the risk of exposing an air of repetition, I would like to pass on these three thoughts which are of prime importance if complete co-operation is to be maintained between the radiologist and the proctologist.

As has been pointed out repeatedly, before an x-ray study is ordered there is a very definite routine from which one should not deviate. This includes a careful history followed by a general physical examination and a thorough proctologic examination.

The latter examination depends upon two pieces of equipment which are always handy, namely, an "educated index finger" and a "seeing eye," augmented by an anoscope and a sigmoidoscope. By the term "educated finger" I refer to a finger which is trained during digital examination to palpate the anal canal and lower rectum and be able to detect differences between the normal and abnormal. By "seeing eye" I mean an observer who knows by sight what normal and abnormal conditions may appear at the distal end of a scope and looks for them.

These steps completed and an x-ray study of the gastrointestinal tract decided upon brings me to my first point: the importance of a proper sequence in the order of study. Due to failure to evaluate symptoms and findings correctly, one quite often sees a low-grade obstructive lesion of the large bowel precipitated into an acute and complete obstruction by administration unnecessarily of our opaque meal by mouth. This is not the fault of the radiologist who is merely carrying out such orders as may have been issued for that particular patient. Proper sequence of procedure in the study should have called for a barium enema first which would permit diagnosis of the lesion without obstructing the proximal bowel. A parallel worth mentioning is the fact that even today castor oil is still ordered in patients with definite symptoms and signs of acute appendicitis.

A second point I wish to make is that regarding proper cleansing of the bowel before barium enema study. Quite frequently the detection of polypi and even some malignant lesions is difficult under the best conditions. We cannot expect the radiologist to give us the best type of x-ray study if we send him patients for barium enema when proper cleansing has not been accomplished.

My third point concerns the importance of shifts of position of the patient so as to obtain

angle shots of the bowel, especially in the sigmoid region, and spot films.

I am in complete accord with both Dr. Green and Dr. Wagner that there is little if any excuse for an x-ray study of rectum, rectosigmoid and perhaps distal sigmoid because in most patients this can be either palpated or visualized. We are, therefore, imposing a useless task on the radiologist and a needless expense on the patient when we request such a study in an area where we can and should make the diagnosis if we will use that educated finger and seeing eye.

Louis J. Hirschman (Detroit, Mich.): I will dispense with the usual compliments to Dr. Green because whatever he does is always very, very good. I just want to emphasize two other points in connection with this admirable presentation. In the first place, one must remember that the roentgenologist is a consultant and it is the close personal relation between the roentgenologist and proctologist that brings the best results to our patients. I don't believe that we should just send a patient to the roentgenologist simply for films. As often as is possible we should be there ourselves so that we can observe under the fluoroscope the different shifts in positions and the uncovering of lesions that are otherwise overlaid by loops of bowel.

For our information as operating proctologists, we should be able to manipulate the sigmoid so that we know how large or how small a sigmoid we have and how adherent it may be, so that subsequently in the removal of carcinoma or other growths we will know whether and how we can place our colostomy and we will also know whether we will be able to have a perineal implantation with good sphincters which we can prognosticate in advance if we know how much movable sigmoid we have.

Again, in these deplorable cases of women particularly who have had previous abdominal operations and who go from one physician to another for the treatment of so-called colitis, if we would manipulate those cases under the fluoroscope after a barium enema, we would find they have distorted, adhered and angulated colons and that the so-called colitis is merely just colonic dysfunction due to pathologic fixation. We would also save many an embarrassment and be able to cure many patients who now are medical and surgical derelicts.

Frank C. Yeomans (Great Neck, N. Y.): I have very little to add, except that I want to compliment the essayist and the other discussers. I want to say that if you do follow the proper routine, of course, which is the digital, then the sigmoidoscopic examination and you do find a pathologic condition, for example, a carcinoma, that doesn't mean that you are not going to have the x-ray examination. It is very essential to have it, of course. The information derived at that time regarding the length and position of the sigmoid and possible detection of other tumors in other situations may be very advantageous in connection with the treatment. It may determine the type of operation that you will perform in a given case.

G. M. Brown (Bay City, Mich.): I should like particularly to ask a question. I am sure we have all had the experience of having reported a negative barium enema x-ray and find in a few months later that our patient has a carcinoma which perhaps has gone too far. When the roentgenologist goes back and looks at the first film, he says, "Oh, there it was. I didn't see it."

I should like to ask how rapidly do carcinomas grow in the different age groups so we can tell how soon to return the patient for a second x-ray if we are not sure of the first.

Richard Brashear (Columbus, Ohio): I would like to ask Dr. Green if these air-contrast films are of the stereoscopic type or whether it is just one film. I have been able to see in a number of these pictures that have been of the stereoscopic type, polypi which I have missed on the single air-contrast film. Occasionally it will give you an idea as to whether the polyp is in the anterior loop or whether it happens to be in the loop that is posterior.

W. W. Green (closing): I am grateful to the discussants for expressing their views on this subject; their remarks have materially contributed to this presentation.

A point that cannot be too strongly emphasized was made by Dr. Taylor when he warned that the use of barium in the presence of a low grade obstructive lesion might precipitate a complete bowel obstruction.

Dr. Wagner has stressed the superiority of the sigmoidoscopic findings over the x-ray examination in diagnosing lesions in the rectal and rectosigmoid area.

I heartily concur in Dr. Hirschman's statement that a close personal relationship between the roentgenologist and proctologist results in better care of our patients. It is a source

of satisfaction to look up during an operation and observe that the roentgenologist has stepped into the operating room to see the correlation between the actual condition and his preoperative findings. Both Dr. Yeomans and Dr. Hirschman brought out a point not made in this paper. They both discussed the usefulness of information gained in fluoroscopy in determining the choice of the contemplated operative procedure.

The time limitations prevent the answering of Dr. Brown's question in its entirety. In those cases in which there is clinical evidence of a lesion not demonstrated by x-ray the latter examination should be repeated every two or three months at the latest until the cause of the symptoms has been discovered.

Dr. Brashear asked if stereoscopic or single films were employed in the air contrast studies. Although only one plate was used for illustration in this presentation, all patients were examined under the fluoroscope and stereoscopic films were taken.

It is exceedingly important to include sigmoidoscopy in diagnostic study of any gastrointestinal problem. This applies to the child as well as to the adult, and should be a routine procedure regardless of the patient's age. . . . If there is an obscure intestinal abnormality, persistent diarrhea or constipation, rectal bleeding, a discharge of pus or mucus, pain, or any other bowel complaint, a sigmoidoscopy should be performed.

TREATMENT OF COMPLETE PROLAPSE OF THE RECTUM

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BEFORE considering the treatment of rectal prolapse, a brief outline of the causes of the disorder should be listed.

It is probable that most men are now in agreement that true rectal prolapse is a sliding hernia of the anterior wall of the rectum, an idea first advanced by Moschcowitz[1] in 1912. This can easily be proved by pressure of the finger on the anterior wall after reduction of the prolapse which will prevent a recurrence, while pressure on the posterior wall allows the rectum to prolapse immediately. It is also noticeable that the lumen of the rectum after prolapse points posteriorly because the prolapsed mass is formed largely at the expense of the anterior rectal wall. Graham[2] gives a good explanation of the mechanics of prolapse. The herniation occurs at the cul-de-sac of Douglas or the rectovesicle pouch and is due to an enlargement of the natural defect in the pelvic fascia that permits the passage of the rectum. The enlargement of this natural defect results from pressure of the contents of the cul-de-sac of Douglas or the rectovesicle pouch into the anterior wall of the rectum, further assisted by such con-

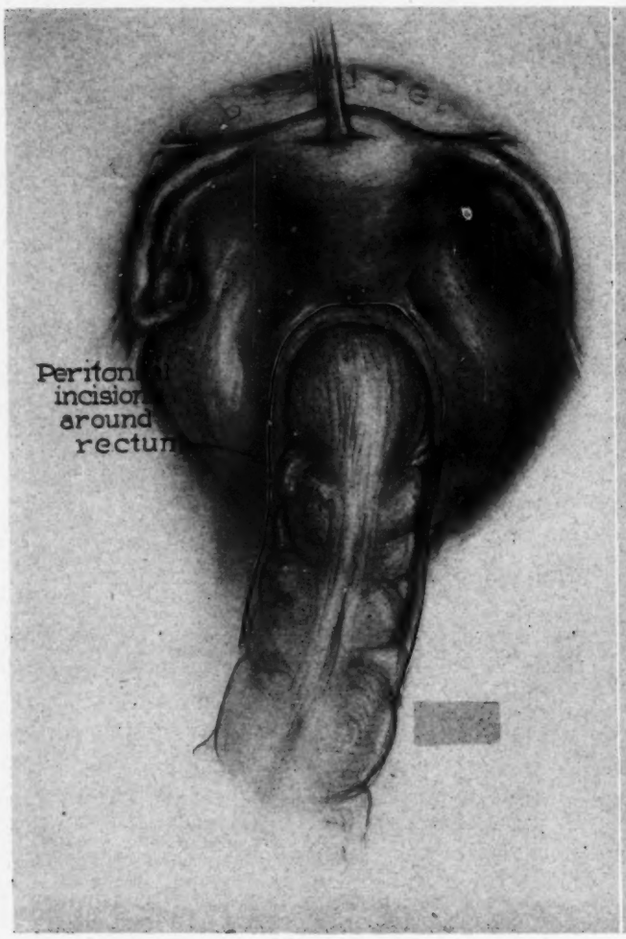

FIG. 1.

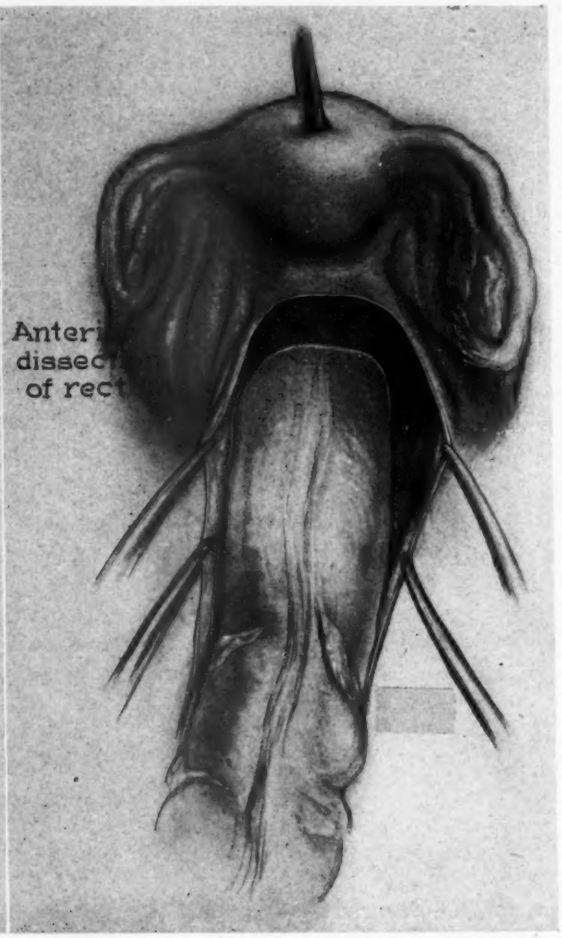

FIG. 2.

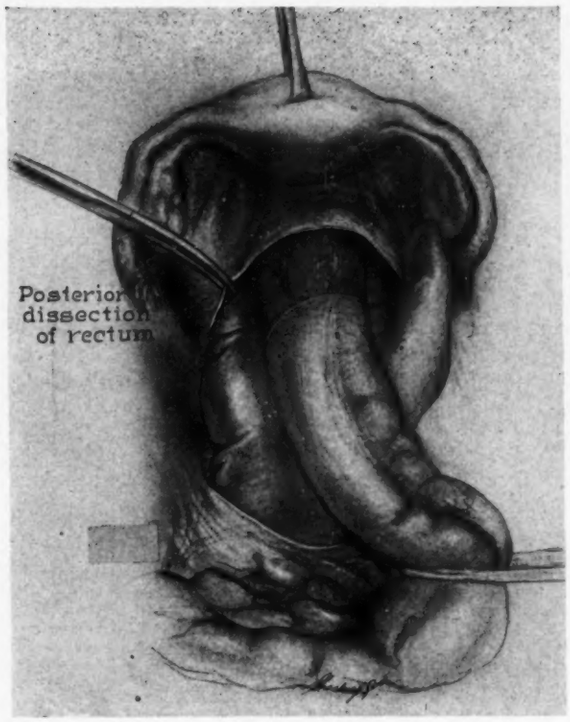

Fig. 3.

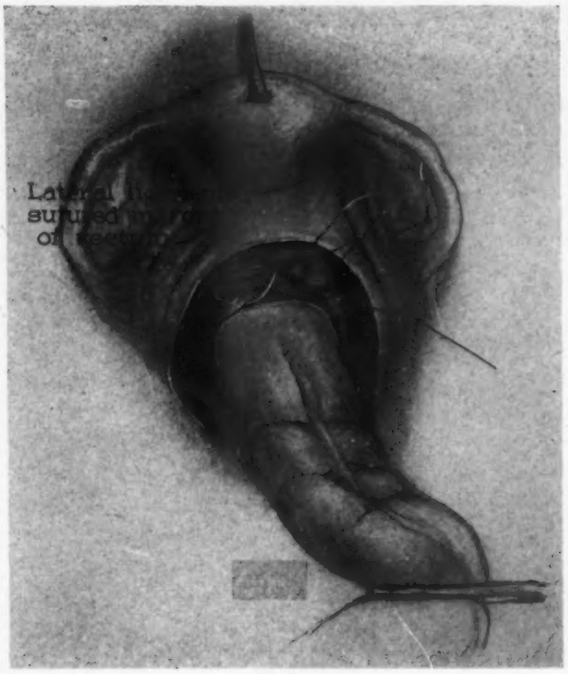

Fig. 4.

tributing factors as general muscular atony associated with old age, lacerated perineum, chronic wasting diseases with loss of perirectal fat, chronic cough or straining at stool or urination.

The pressure into the anterior rectal wall gradually separates the levators ani, thus permitting enough anterior wall of the rectum to be invaginated into the lumen of the rectum that the latter protrudes through the anal canal. The normal angulation of the rectum becomes obliterated, the posterior wall of the rectum is carried forward from the hollow of the sacrum and the fascial supports become most inefficient due to the stretching. Over the years the mesentery of the small bowel becomes elongated, permitting the latter to lie in the hernial sac and this accounts for the tympanic note on percussing the prolapse.

Several surgical procedures have been advocated for the cure of rectal prolapse. Among these are injection of sclerosing agents, operations designed to narrow the anus and rectum, operations to suspend or fix the prolapsed bowel, operations to restore the pelvic floor, those to resect the prolapsed bowel and others to obliterate the pelvic cul-de-sac. We think that injections of sclerosing agents are sufficient in the cases of smaller recurring rectal prolapse of one or two inches. For the other cases of large recurring prolapse we agree with Graham that adequate treatment should be removal of the sac and repair of the anatomic defect in the pelvic fascia, together with additional fixation of the sigmoid colon.

We should like to report nine cases of rectal prolapse seen over a period of as many years. The first three were patients with acute massive prolapse for which no treatment was necessary other than the simple reduction of the prolapse. In each instance it was the first such attack and there was no recurrence. Each patient was a white woman in the fourth decade. One was straining to urinate after eating watermelon and the other two were straining at stool. A good procedure to follow in such cases is to use liberal quantities of toilet tissue when manipulating the mass. Otherwise the slippery surface of a mass the size of a football is most difficult to handle.

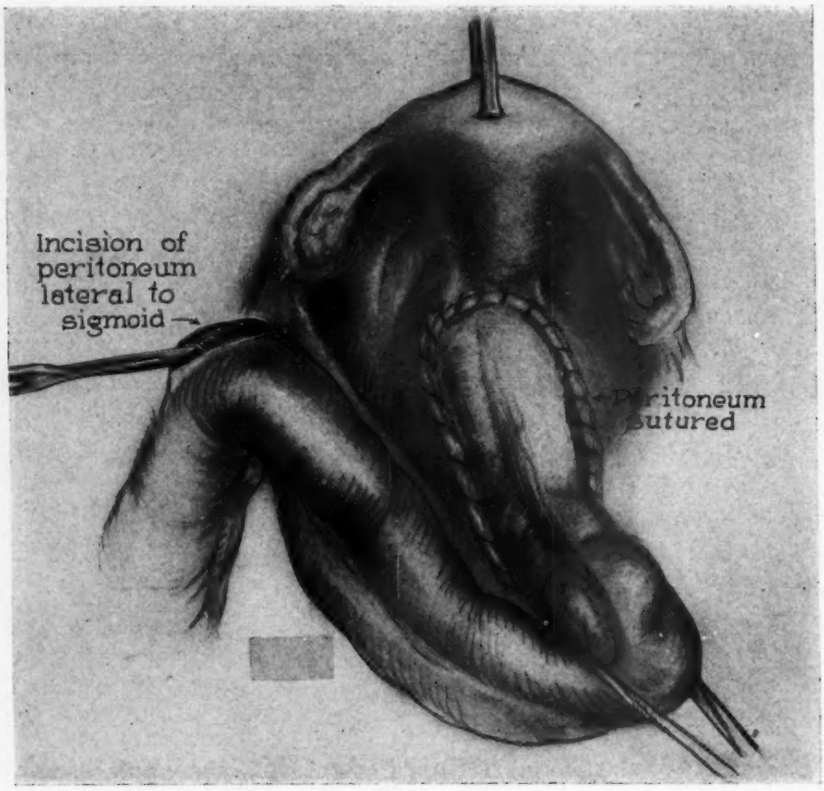

Fig. 5.

Of the remaining six patients, three were injected with sclerosing agents. One boy, age two, was injected with 3 cc. of ethyl alcohol on each side of the rectum above the levators. This was a failure. The other two were white women, thirty-nine and sixty-six respectively who were injected with 1 per cent phenol in castor oil above the levators at each side and posteriorly. Three ounces were injected at each side and one ounce posteriorly. Then, about one ounce of 5 per cent phenol in olive oil was injected under the rectal mucosa, spotting about 5 cc in each of about six sites. Both patients obtained a cure.

The remaining three patients were subjected to abdominal operation. The first was a Negro boy of twenty-two who had a cogenital prolapse of five to 6 inches at each stool. At operation it was found that the rectum was edematous, leathery and thickened so that it filled the entire pelvis. The perirectal tissue was tough, inelastic and edematous and it was technically impossible to properly free the rectum from the sacrum or to expose the pelvic fascia over the levators. All we could do was bring the peritoneum under the rectum as a new pelvic floor. He obtained a partial cure in that only about 2 inches prolapsed after operation.

The last two patients were operated on after the technic of Graham, together with construction of a new bed for the sigmoid which we have added. After a spinal anesthetic an ordinary rectal tube was inserted in the rectum for about 2 inches before the abdomen was opened. Beginning at about the level of the superior hemorrhoidal vessels, the peritoneum on each side of the rectum was incised down to the level of the cul-de-sac and then completely across the cul-de-sac. Finger dissection then freed the rectum from the hollow of the sacrum down to the coccyx just as though removal of the rectum were contemplated. Blunt dissec-

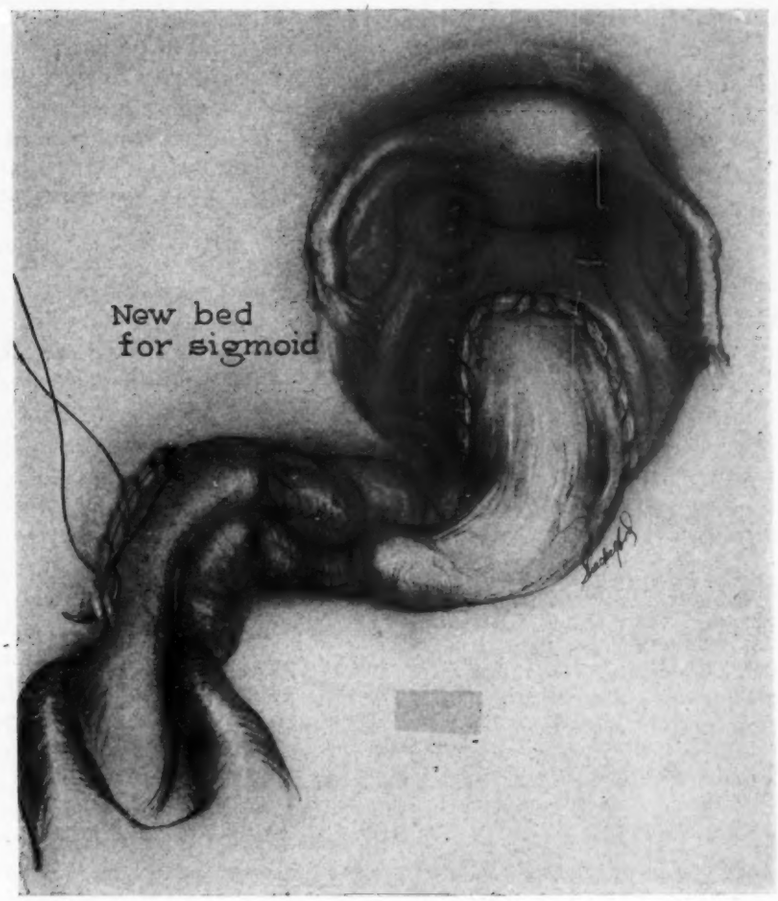

Fig. 6.

tion then freed the rectum anteriorly. The defect between the levators could easily be felt. At this point Graham closed the defect by approximating the levators with sutures through the pelvic fascia over the levators. Both of our patients were males in whom we found this procedure technically impossible. Instead of suturing the levators together we brought the lateral ligaments of the rectum across in front of the rectum and sutured these ligaments together with one or two linen sutures. This took up the slack in the overstretched lateral ligaments and prevented the anterior wall of the rectum from prolapsing through the defect between the levators ani. Then, with the rectum held up taut the cut edges of pelvic peritoneum were turned under and sutured to the rectum at the sides and anteriorly. This obliterated the cul-de-sac of Douglas or the rectovesicle pouch entirely. As a further support to the repair we made a new bed for the sigmoid by incising the lateral peritoneal leaf from the brim of the pelvis up the lateral gutter for a distance of 4 to 5 inches. After blunt dissection of the thin areola tissue under the sigmoid, the peritoneal leaf was sutured to the wall of the sigmoid while the latter was held up taut. Before closing the abdomen the rectal tube, previously inserted, was pushed up into the rectum and sigmoid by an attendant while being guided by the surgeon's hand. The abdomen was then closed and the protruding end of the rectal tube sutured to the skin over the inner side of the gluteal region. The tube remained for about three days when gas began to pass. By this time the patient was taking liquids by mouth. Mineral oil was then started, one ounce twice a day, the diet increased and the bowels began moving on the fourth

or fifth day. Complete cures resulted in these last two patients and we think that the above procedure is the operation of choice for patients with massive rectal prolapse. The new support of the anterior rectal wall made by suturing the lateral ligaments together in front of the rectum, the obliteration of the pelvic cul-de-sac, and the scar tissue that forms around the rectum in its new bed and the sigmoid in its new bed makes a recurrence virtually impossible.

In none of these patients has it been necessary to tighten or do any plastic work on the sphincter muscles since in each case the muscles were competent.

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DISCUSSION

Thomas Brockman (Greenville, S. C.): A truly significant contribution to the field of geriatrics was made by the late Dr. Granville S. Hanes of Louisville, Ky. Many will remember Dr. Hanes although few were taught his technic. I watched him employ his technic on several patients. Among his patients was one of our own members who, as a result of a Whitehead operation, suffered from a partial prolapse or sagging of the rectal mucosa. Dr. Hanes' technics for treatment of complete and partial prolapse has proved highly successful.

I should like to cite my own experiences with Dr. Hanes' methods. Most of my patients were elderly and their muscular systems had become relaxed, or atonic, as we call it. The majority had borne one or more children. For the most part they were as healthy as may be expected of eighty to eighty-six-year-old women. The difficulty was that when they stood erect for a short period, the anal canal muscles and the rectal muscles would relax and a tubular or globular mass about the size of an ordinary water glass would protrude the entire anal canal and rectum. Naturally, the patients were seriously handicapped even for simple household duties and exercise, such as collecting eggs, feeding chickens, etc. The patients had been remarkably robust; one was still riding horseback at the age of seventy-five, most of the riding being over the prairies and oil fields of Mexico. It is one of the few gratifications in a physician's practice to encounter such fine patients.

The technic of treatment is somewhat as follows: The patient is placed in a prone position on the operating table with pillows and sand bags under the midriff. The buttocks are retracted by adhesive strips placed on either side. The perianal area is prepared as for surgery by swabbing with merthiolate. The only anesthetic is a very light skin dose of 1 per cent novocain, injected at needle point on two sides. Several ounces of warm, sterilized castor oil is injected perianally. This requires a large No. 16 needle which is introduced along the side of the rectum by guiding its course in the rectum by a gloved finger. A pool of oil is injected on either side or laterally until the surgeon can feel the bulging warm oil mass. The average dose is 90 to 100 cc. of castor oil.

Stimulants are given during the operation and occasionally gas is required. Vigorous finger massage is instituted promptly while the patient is on the table. Heat and massage are continued for one to two hours each day for several days to prevent pooling of the oil and adhesive strips are applied constantly.

The patient requires hospitalization and full time nursing care. Slight shock may be combatted by the usual methods, chiefly stimulants of coramine, oxygen with the rare use of demerol or a very mild hypodermic of pantopon. The chief weapons for after-care are heat and massage and in the later stages the patient appreciates hot sitz baths.

I have never encountered a recurrence. The age of the patient may be a factor in this observation. Nevertheless, the remaining years of the elderly woman are made comfortable and happy.

Rufus C. Alley (Lexington, Ky.): It is a pleasure to discuss such a scientific and well presented paper.

The multiplicity of treatments, operative and otherwise, which have been advocated for rectal prolapse indicate that no single method is ideal or universally applicable. This point is illustrated by the fact that Drs. Hayes and Burr used three different methods in the treatment of

nine patients: (1) simple reduction; (2) sclerosing injections and (3) surgical repair.

Their explanation of the mechanism of complete prolapse is perfectly logical as is their method of surgical correction of the responsible anatomic defect.

The great majority of patients with rectal prolapse that I see are either young children or elderly debilitated individuals. In my opinion a child with rectal prolapse, either partial or complete, should not be subjected to surgery until all simpler corrective procedures have been tried and have failed. Co-operative management by proctologist and pediatrician will, in most instances, lead to spontaneous cure in these cases. The reason for this is because as the child grows the rectal bed increases in length more rapidly than does the rectum, thus taking up the slack which was responsible for the prolapse.

The elderly debilitated patient with rectal prolapse is an entirely different problem. In most of these patients spontaneous improvement is not to be expected and because they are poor surgical risks major surgery is not feasible. In this type of patient the judicious use of sclerosing injections is the safest procedure for palliation.

E. G. Martin (Detroit, Mich.): I would like to restrict my discussion to the paper as presented which I enjoyed very much. I would like to accentuate what the Doctor has reported, that after he has done what he has disclosed in his drawings, the patient will not have a prolapse again; it would be practically impossible.

The first thing that happens is prolapse of the sigmoid into the rectum, a colonic rectal prolapse. This has been classified over the years as third degree. Since this is the first thing that happens, I classify it as first degree.

Patients who have their two ligaments, one of which is at the junction of the pelvic colon and rectum and the other at the pelvic colon and iliac colon, can have prolapse if those ligaments are not normal. I do not know about the defect of the anterior part, but I do know from rather long experience in quite a number of patients that if you put your hand in the abdomen when you have it opened and pull up the gut, it will not recur as long as you hold your hand there. It cannot recur, because it cannot get away from your hand. We simply fasten the gut to the iliac fascia or the psoras spina muscle, and about five or six plain catgut sutures cover up the raw end of it and we can omit all of this dissection and prolongation of this operation which Dr. Burr has so well presented. Another prolapse probably will not occur after the gut has been well fastened; at least, in our experience we have never seen or heard of a recurrence of prolapse after this treatment.

Emmett H. Terrell (Richmond, Va.): I would like to discuss rather briefly the method which we use in the treatment of prolapse in children. A distinction should be made between simple incomplete and complete varieties of prolapse of the rectum. In children almost invariably it is simple prolapse of the mucous membrane.

Prolapses occurring in children are not necessarily confined to those that are debilitated but we find it very frequently in well nourished and otherwise perfectly healthy children. For the last twenty-five or thirty years, we have simply been treating these prolapses in children by the injection of quinine and urea hydrochloride, either 2 or 3 per cent, after the same method that you use in the treatment of simple, uncomplicated hemorrhoids. I cannot say how many children we have treated during this period, but it has been quite a large number. In most instances the symptoms are entirely relieved after the first treatment. In a few cases when you have had to repeat the treatment for as many as four or five times, there has not been a failure to completely relieve the symptoms. As far as I know, there have been no recurrences, after this method of treatment. Of course, it would not apply if the muscular parts of the bowel were prolapsed. This treatment is only applicable in those cases in which the mucous membrane protrudes.

Rudolph V. Gorsch (Flushing, N. Y.): A great deal has been written about injection therapy in treating prolapse. Obviously, it is not always a satisfactory method. There is a very important technical point I would like to bring out about it. I remember years ago we brought Dr. Hayes up to New York to see how he conducted the injection method. Having used it beforehand, I learned very forcibly at that time that we were not using the proper technic. In using the injection method, whether you use quinine urea, castor oil or hydrochloric acid, it is essential that you put your solution directly along the inner edge of the bowel wall because I think ultimately your success upon

holding up the prolapse depends upon the amount of fibrosis you get. You absolutely have to flood the solution right alongside the bowel wall. If you put it out a centimeter or a centimeter or two, obviously you don't get any fibrosis or fibrous fasciculi which will eventually hold the bowel wall up.

I think that is a very important point in the injection treatment.

Raymond L. Murdoch (Oklahoma City, Okla.): There is a great tendency when a paper has been published, particularly one with excellent diagrams, as Moschcowitz paper had, subsequently to say that all prolapses are caused by defect in the fascia of the anterior wall and that the anterior wall of the rectum is the part that is the initial part of the prolapse. But I do not believe that all prolapses that appear on our hospital records are prolapses through the cul-de-sac. I feel that the great majority of them are just a little more severe or extensive processes of ordinary hemorrhoids, the prolapsing of large hemorrhoids with a little additional severity of that process and then diagnosis in hospital records appears as prolapse. So the thing that must be done in every case is to feel the prolapse all around as well as look at it and percuss it, in order to detect a tympanic note although I have found this very difficult to elicit. I have been very much in doubt as a matter of fact whether only the mucous membrane prolapsed or whether all coats of the bowel prolapsed in the patients I have had.

Therefore, I think it important not merely to accept Moschcowitz's beautiful diagrams, but to take each individual case, inspect it very carefully and particularly feel it.

The great majority of patients who are diagnosed as having prolapse show a mass out at the anus. This is a prolapse of the anus rather than a prolapse of the rectum. Next time determine the amount of sulcus. Start in at the anal border and see whether the finger will extend into a fairly normal anus before it strikes this intussuscepting mass. In other words, see how much sulcus there is there before you get the prolapse.

The next method is the Moschcowitz diagram procedure. In females it is unnecessary to go into the abdomen to remove and correct the prolapse. The abnormal mass can be removed by going through the rectal vaginal septum. It is a common practice for correcting defects there.

The very rare case is the one in which it is necessary to go into the abdomen to make a fixation of the colon. If this canal is narrowed and the defect repaired which can always be done in the female from below, then it will seldom be required to go into the abdomen for operative procedures on prolapse in females.

Harry B. Burr (closing): I would like to thank the discussers for their kind and generous remarks. It is a healthy thing to have a difference of opinion.

I agree with Dr. Alley, of course, that in treating old people, one should not subject them to abdominal operations. I do not believe in removing a prolapse of the bowel from below. I think it is dangerous because frequently loops of small bowel are in the prolapsed mass. I am speaking of the massive prolapse, not the small prolapse of the mucous membrane.

DIAGNOSIS AND TREATMENT OF PAPILLARY ADENOMAS OF THE RECTUM

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New York, New York

THE most common tumors found in the rectum are those which arise from the mucosal glands and are classified as adenomatous tumors. These tumors in accordance with the histologic appearance may be divided into two main groups, benign and malignant. Benign tumors, regardless of shape and size, are often termed polyps, while the malignant variety are known as malignant polyps or rectal cancer. Differentiation between the benign and malignant group, with recognition of the characteristics of each, is essential if patients are to receive the most appropriate treatment.

There are three clinical types of benign adenomatous tumors: (1) areas of hyperplasia, (2) adenomas and (3) papillary adenomas.

Areas of hyperplasia are small, sessile, slightly raised tumors which occur most frequently in the mucosa above or below rectal cancer and also on mucosa which has been exposed to irritation for a comparatively long period, e.g., the surface of a colostomy. It is possible that areas of hyperplasia are at times forerunners of more mature adenomatous tumors.

Microscopically, areas of hyperplasia show only slight differentiation from the surrounding mucosa. The glands are somewhat larger and contain cells which are slightly elongated and more closely packed. The nuclei may be hyperchromatic. However, the orderly arrangement of the mucosal glands is nowhere lost and blood-vessel distribution is identical with the adjacent normal areas.

Adenomas are the most common of the three benign varieties. These polyps are usually small or of medium size. They may be single or multiple, smooth or ulcerated, sessile or attached by a pedicle. The induration is usually sufficient for recognition by palpation with the finger.

Adenomas are composed of arborescent masses of large proliferative glands upon an orderly frame-work of connective tissue which radiate from a central fibrous stalk in which the blood supply is carried and which is developed from the submucosa. The stalk may be elongated to form a pedicle, in which case it is covered by normal appearing mucosa. The glands are much larger than normal, both in length and diameter, and vary in degree of mucus secretion. The cells of the glands are generally closely packed and elongated, and their nuclei are compressed and hyperchromatic. Mitoses are frequently seen and the cells are sometimes multilayered.

Papillary adenomas are seen less commonly than the other two groups. They generally occur in an older age group than the adenomas and are usually single and sessile; when they are large, they have overhanging margins. A pedicle, if present, may be almost equal in diameter to the tumor itself. The base of the polyp is frequently circular but often becomes quite irregular as the size of the polyp increases. The thickness varies widely in different cases and also in the same tumor. One area of the tumor may be a millimeter while another area of the same tumor is several centimeters in thickness. The surface presents a papillary or in some cases a lobulated appearance which is characteristic of this type of disease. Papillary adenomas are soft and spongy and it is not unusual for them to be missed on rectal palpation.

Papillary adenomas are similar in microscopic appearance to adenomas; in addition the greater part of the surface is composed

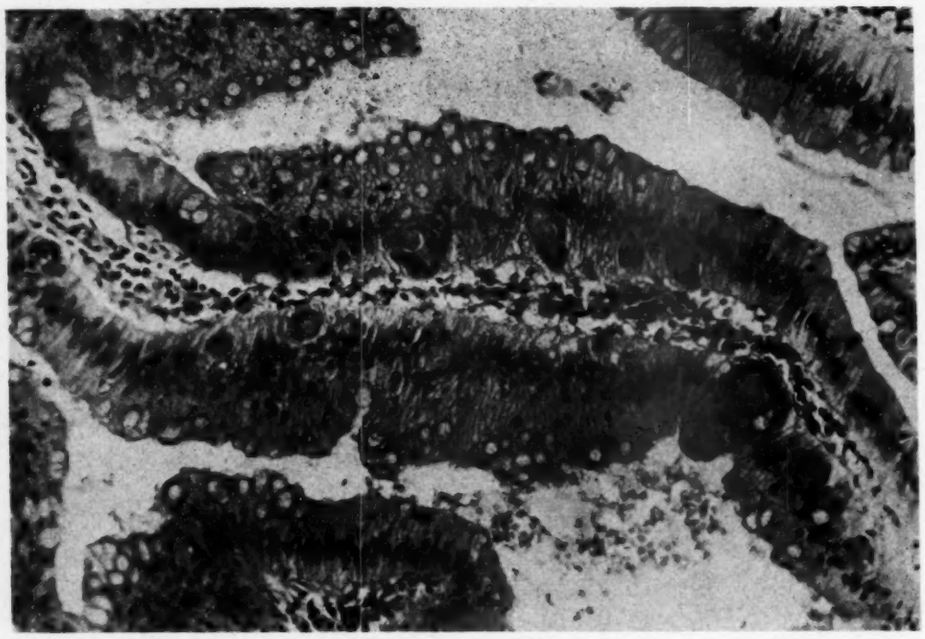

FIG. 1. Shows early secondary gland formation or atypical change in a villus.

of thin villous processes covered by single or multiple layers of elongated columnar cells upon a basement membrane supported by a delicate fibrous tissue core which carries the blood supply. Small papillary adenomas develop from a single central fibrous stalk, but the majority of them are composed of multiple stalks springing sometimes from a wide area, each stalk supporting a papillary aborization. Infiltration of eosinophiles, plasma cells and lymphocytes are frequently seen in the central stalks and their prolongations. Also, it is not uncommon to find interstitial edema of the individual villous process. The degree of mucous secretion can vary markedly in different areas of the same tumor. Mitoses are frequent and not considered significant.

Atypical changes may be present in the papillary adenomas, with or without areas of outright malignant transformation. These changes consist of focal or diffuse areas of papillary tufting of epithelial cells into the lumen of the glands or onto the surface of the villi without disruption of the basement membrane. In some cases there may be diffuse atypy throughout the specimen. In others, there is a relatively benign structure throughout the greater part of the specimen, with focal malignant areas without infiltrating qualities, as evidenced by distortion of gland structures and divergence in size and shape of the nuclei with apolarity. Others will show a diffuse atypical structure with small focal areas of frank carcinoma infiltrating within the polyp. Many degenerative changes simulating malignancy may be seen in the tips of the villi. Also, secondary villi may be found springing from within the lumen of glands.

Papillary adenomas may be separated for convenience according to their clinical and microscopic appearance into four groups: (1) tumors that are clinically and microscopically benign; (2) tumors which show microscopic atypical change; (3) papillary adenomas in which there are islands of adenocarcinoma and (4) fully developed infiltrating carcinoma in which areas of benign papillary adenoma still remain.

One cannot overemphasize the importance of recognizing clinically the characteristics of the benign, atypical and malignant types of papillary adenomas. Classification may be impossible by any

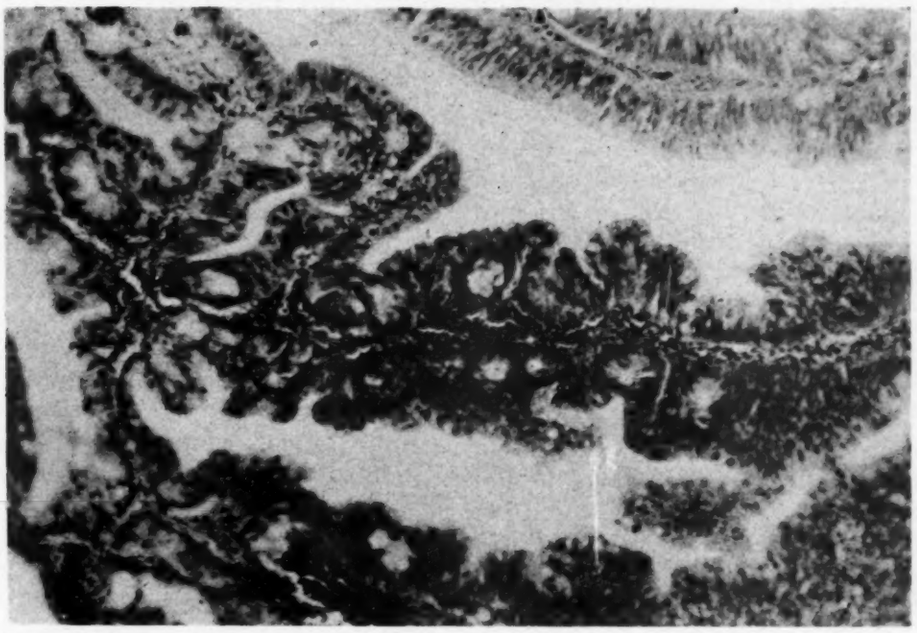

FIG. 2. Shows more advanced secondary gland formation or atypical change in a villus with moderate papillary tufting of surface epithelium.

other method prior to complete surgical removal. Due to the previously mentioned wide microscopic variations, repeated biopsies often fail to reveal existing atypical or malignant features. Factors to be considered in estimating malignant probabilities are induration, ulceration, color and bleeding or oozing following moderate trauma to the polyp.

The small or medium-sized purely benign lesions are soft and about the same color as the adjacent mucosa. They are not ulcerated and do not bleed readily. Benign polyps revealing atypical changes will show a slightly increased vascular appearance to the above, bleed or ooze slightly to trauma. All polyps which have undergone definite malignant change have a recognizable degree of duration and sufficient blood supply to produce considerable oozing or bleeding when traumatized and after taking a biopsy. It is not unusual to find superficial ulceration in a percentage of malignant cases.

Determination of the appropriate form of treatment is at times a difficult problem. Factors worthy of greatest consideration are the previously described clinical characteristics, together with the size of base and extent of involvement of the rectal wall, and the presence or absence of induration at the base. The objective in all cases is complete eradication of the tumor with a minimum amount of morbidity and rectal deformity.

For descriptive purposes treatment may be divided into steps or stages: A number of the smaller papillary adenomas are eradicated by the initial procedure while the more extensive adenomas require additional treatment. Partial or complete removal of all papillomatous tissue is attempted in the first stage. Removal may be by means of an electric snare or electric snare together with fulguration of the base of the polyp. In certain cases local surgical removal by dissection and suture is the method of choice. Methods which permit histologic study of the tumor tissue removed are preferable to those which destroy it.

The second step of treatment consists of radiation therapy. It is reserved for patients in whom snare and fulguration or local surgical removal have failed to eradicate the disease completely. Radiation

therapy consists of external applications of high voltage roentgen rays, surface applications of radium and interstitial implantations of gold-filtered radon seeds.

External applications of high voltage roentgen rays are administered through six or seven pelvic portals, the rays being focused at the center of the lesion. Surface applications of radium are administered by means of a rectal applicator which holds the radium at a distance of 10. to 1.5 cm. from the surface of the polyp. Small daily dosages delivered in this manner produce a favorable effect upon the tumor without causing any unfavorable reaction to the patient. Islands of polypoid tissue which remain after moderate dosages of radium have been administered by rectal applicator, may be treated by implantation of gold filtered radon seeds. It is our impression that snare removal, superficial fulguration and radiation therapy is followed by less deformity of the rectum than that following focal surgical removal of a large sessile tumor.

Radical surgical removal of the rectum constitutes the third step of treatment. This radical treatment is reserved for those conditions which have not been cured or controlled to a satisfactory degree by the above less radical methods. Tumors which show induration at the base and those with fully developed papillary adenocarcinoma should be treated in this manner.

All patients who have received conservative treatment for papillary adenomas warrant careful post-treatment observation. There is marked tendency for these tumors to recur. Recurrence may be either early or late. Moreover, even with an initially benign form of tumor, recurrences may reveal atypical or malignant cells. Because of the frequency of carcinomatous change in the primary and recurrent tumors, all patients must be carefully watched and the lesions considered to be potentially malignant.

DIAGNOSIS AND TREATMENT OF MUCOSAL POLYPS OF THE RECTUM AND COLON, WITH EARLY MALIGNANT CHANGE*

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THERE are certain conclusions which are generally accepted at the present time concerning mucosal polyps of the rectum and colon: (1) Polyps of the terminal bowel, the majority of which are adenomatous in character, are true tumors and not the result of inflammatory processes. Clinically, they must be considered premalignant lesions.[1] (2) The incidence of mucosal polyps in the adult population is much greater than has previously been suspected. Haug[2] and I in a series of 1,843 autopsies performed at the Pathological Laboratory of the New England Deaconess Hospital found 311 patients with benign mucosal polyps, an incidence of 7 per cent. Forty-two per cent of these patients had multiple lesions. Helwig[3] more recently has reported an incidence of 9.5 per cent in a large autopsy series. (3) The detection of large numbers of colon and rectal polyps has been difficult. Only a small percentage of such patients complain of rectal bleeding, and a still smaller group is found to have polyps which are benign and yet have attained sufficient size to cause obstructive or other bowel symptoms.

It has been demonstrated that over 50 per cent of the benign polyps of the terminal bowel are within reach of the 10-inch sigmoidoscope,[2] but the radiologic detection of small tumors beyond the reach of the sigmoidoscope is unsatisfactory. Only a small number of the mucosal polyps of the right colon, transverse colon, descending colon and upper sigmoid, which we believe to be present, are detected by the usual barium enema radiographic studies of the colon or by the contrast air technic. Because of the high percentage of these tumors within reach of the 10-inch instrument, however, as we have broadened our indications for sigmoidoscopic examinations we have discovered more and more mucosal polyps.

To discover the greatest possible number of premalignant lesions of the colon and rectum, a sigmoidoscopic examination should be carried out on all patients over thirty-five years of age as a part of a general physical examination. Internists, surgeons and all physicians doing general diagnostic work should be prepared to do examinations of this nature. It requires a minimum of experience to learn to pass a sigmoidoscope and to determine the presence or absence of a tumor. Once a tumor has been discovered, however, it may require the highest degree of skill and experience to determine the presence, degree and extent of any malignant change which may be present and the type of treatment indicated.

It is the purpose in this presentation to review the criteria for the diagnosis of cancer in colon and rectal polyps, and to discuss the treatment indicated when malignancy is found. Also, a series of twenty-two cases will be reported in which the diagnosis of malignancy was made sufficiently early so that local excision or destruction of the polyp was adequate to control the disease.

This series of twenty-two patients is admittedly a small number as compared with the number of patients seen at the clinic with cancer and benign tumors of the colon and rectum. Over 2,000 patients have now

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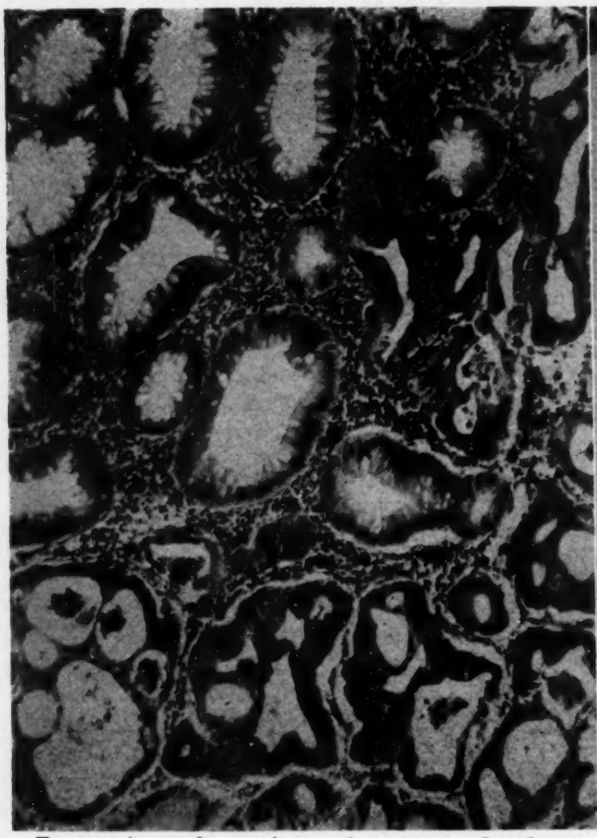

FIG. 1. An early carcinoma in a mucosal polyp; tumor removal by local excision. × 150.

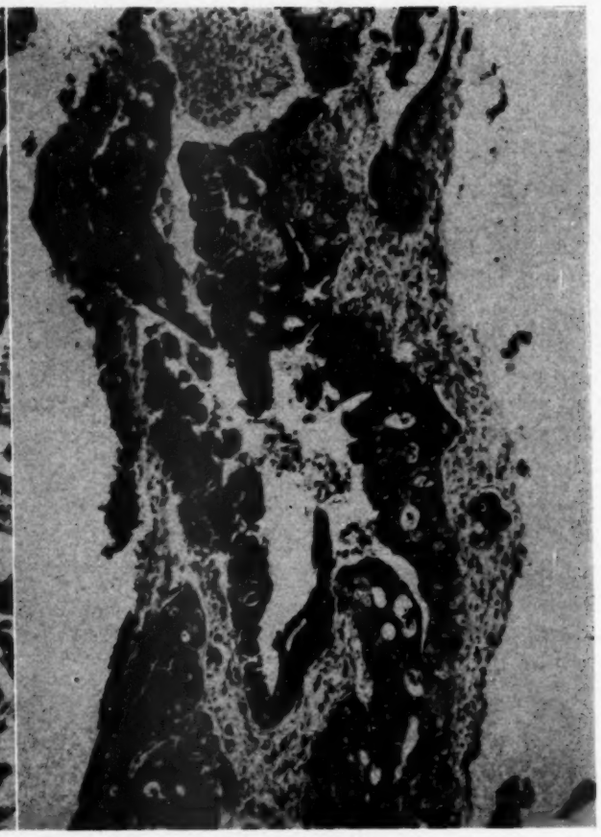

FIG. 2. A small focus of carcinoma in a mucosal polyp; tumor removed by local excision. × 150.

been operated upon for malignancy of the large bowel and several hundred additional patients with benign polyps of the colon and rectum. Seventy-six histologic specimens of benign polyps of the colon and 684 of the rectum have been studied in addition to the large number of patients who have had small mucosal polyps fulgurated through a sigmoidoscope without biopsy specimens being removed. As more and more polyps are discovered, the problem of the early diagnosis of malignant change in such tumors with the possibility of avoiding radical resection of the bowel in a small number of such patients will become increasingly significant.

In the detection of malignancy the value of palpation of these tumors cannot be overemphasized. The soft, pulpy, nonindurated, freely movable polyp discovered in the ampulla of the rectum will rarely be found to be cancer. Induration, fixation and ulceration to the examining finger, however, are almost always pathognomonic of malignancy. The visualization of these tumors is less important than palpation or histologic study. To the trained observer, ulceration and defects in the usual normal-appearing mucosal surface of these tumors are significant. The typical ulcer of malignant disease, with firm, rolled edges is diagnostic.

The final proof of malignant change in a polyp depends on histologic study, not only of biopsy specimens of the polyp but of representative sections of the entire tumor, together with its stalk and attachment to the bowel wall as malignant change may develop in any portion. It has not been an infrequent experience to have suspected cancer in a polyp because of firmness or induration to palpation and only after a large number of biopsy specimens have been taken from various sections of the tumor could malignancy be demonstrated.

Histologically, it may be relatively difficult to determine the presence or absence of malignant change in many intestinal polyps. Frequently patients are referred to the clinic for a final diagnosis where our pathologists disagree with previously reported findings. It is essential in these borderline cases that a pathologist experienced in this field be available. Warren[1] and I in a paper on this subject have stated that "If we accept three important criteria of malignancy—anaplasia, irregularity of architecture and invasion—it is necessary to have at least two of these three factors present before making a diagnosis of malignancy. It is possible for any one of these three criteria to be present without actual malignancy, with one exception. Definite lymphatic or intravascular invasion means clinical cancer."

These criteria from a histologic standpoint have been followed in our patients for a number of years and have been highly accurate in the determination of actual cancer.

When palpation reveals a freely movable, soft, non-indurated tumor and it is possible to demonstrate histologically that there is no malignancy in any section of a polyp, its stalk or adjacent bowel wall, local excision or complete fulguration is indicated. These patients should be studied for additional polyps because of the high frequency of the multiplicity of these tumors and, probably because of the inherent growth defect in these patients, they should be examined at intervals indefinitely.

When palpation of such a tumor reveals induration, ulceration and fixation of the tumor and a biopsy specimen taken from the periphery shows malignant change, radical resection is the only procedure indicated.

When palpation reveals induration, ulceration and fixation of a polyp and such a biopsy does not reveal malignant disease, the final diagnosis should be deferred until repeated histologic studies have been made of all sections of the tumor together with its stalk and adjacent bowel wall; and only then, with such histologic findings interpreted by an experienced pathologist, can cancer be ruled out.

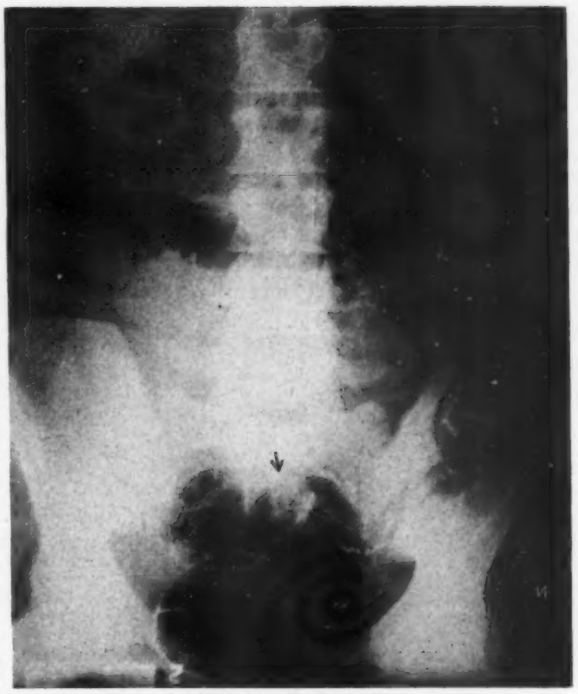

Fig. 3. Multiple polyps in sigmoid area.

When palpation reveals a freely movable, soft, non-indurated tumor and histologic studies disclose a polyp with early malignant change confined to a single area and not involving the stalk or adjacent bowel wall, then, and only then, should local excision or destruction of the tumor be considered.

In a review of the twenty-two cases reported in this series, certain additional data are of significance. Twelve of the patients who were treated by local excision or destruction of the tumor had polyps showing local, early malignant change within reach of a 10-inch sigmoidoscope. Two patients had a subsequent radical resection because of a suspected involvement of adjacent tissue by the malignant process but no malignant change was found in the removed specimens. Bleeding was noted by all of these patients. Age and sex factors were not significant.

Ten of these patients had tumors in the colon, eight in the sigmoid area and two in the descending colon. Forty-one patients

FIG. 4. Large benign mucosal polyps in sigmoid area.

FIG. 5. Large fecalith mistaken for tumor.

have been operated upon at the clinic by colotomy with local excision of the polyp. In this group, histologic study revealed no malignant change in thirty-one patients; in twenty-eight the polyp was in the sigmoid area, in one patient in the transverse colon and in two patients in the descending colon. Ten patients had polyps with localized early malignant changes; eight were in the sigmoid area and two were in the descending colon. Two of the patients with early malignant polyps later had resection because of the extent of the tumor but no residual cancer was found. Bleeding had been noted by thirty-eight of these forty-one patients. There was one postoperative death, a mortality of 2.4 per cent.

It is to be noted that the incidence of early malignant change in those polyps found above the reach of the 10-inch instrument is many times greater than in those found below this level. The incidence in this series was 24 per cent. This finding is probably explained by the fact that few tumors are discovered in the colon until they have reached a centimeter or more in diameter, have been present for an appreciable period of time and may have developed peripheral ulceration with bleeding resulting from their beginning malignant change, which symptom originated the investigation during which the tumor was discovered.

In conclusion, twenty-two patients are reported with mucosal polyps of the rectum and colon having early malignant change, treated by local excision, and followed for varying intervals of time up to seven years with no recurrences. It is to be emphasized that no attempt is made in this presentation to advocate anything but the most radical type of surgical procedure for clinical cancer of the colon and rectum. It is to be hoped, however, that as the medical profession becomes increasingly aware of the significance and frequency of the premalignant lesions of the colon and rectum, not only will an increasing number

of patients be prevented from developing cancer of the terminal bowel by the discovery and destruction of these tumors in this premalignant state but there will be an appreciable number of patients having polyps showing early malignant change who may be safely treated conservatively.

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DISCUSSION OF PAPERS OF DR. BINKLEY, DR. SUNDERLAND AND DR. SWINTON

D. R. Laird (Portland, Ore.): It is a pleasure to discuss these interesting papers but difficult in that they are so complete and there is so much agreement in the main.

The division of benign adenopapillomatous tumors into three clinical types with correlation of the clinical characteristics and histologic appearance is of value and I agree entirely with Dr. Binkley's observations.

It should be emphasized again that the clinical characteristics mentioned by Dr. Binkley, i.e., induration, ulceration, fixation and vascularity certainly are as important as the microscopic findings of biopsy in determining the type of treatment. I agree that even multiple biopsies may fail to reveal the presence of malignancy and that when possible the entire lesion should be removed in one operation so that complete histologic study may be made.

Dr. Binkley's division of papillary adenomata into four groups for the purpose of clarifying treatment is advantageous. There is no doubt that the decision as to what type of treatment is to be used is at times difficult and should be made by one having great interest and experience in this field. So many variables are present that hard, fast rules cannot be made to cover individual situations. As a guide we should all ask ourselves this question when confronted with such a decision, "What would I choose to have done if I were the patient"? This helps to bring the problem home in a real way.

In regard to treatment of these lesions, fulguration, snare excision and local excisions to remove the entire thickness of the rectal wall all have their place in clinically benign to border line lesions. The principle of fitting the operation to the patient and not the patient to the operation pay dividends in reduced mortality and morbidity. This is illustrated by the unusual circumstances involving a patient in which two previous attempts elsewhere, over a two-year-period at destruction of adenopapillomatous hyperplasia of the entire rectal ampulla, had failed. When seen, the patient was adamant in refusing any type of abdominal operation. The lower 10 cm. of her rectal mucosa was diffusely involved by adenomatous hyperplasia varying from 1 to 3 cm. in thickness. Fulguration was considered hesitatingly because of the huge area involved but, when it was discovered that the rectosigmoid was mobile and could be brought down to the anus easily, the entire rectal ampulla, wall and all was dissected free and the rectosigmoid sutured to the sphincters. This patient left the hospital on the eighth postoperative day with excellent bowel habit and control and has remained so for the past eighteen months. This same procedure has been used on three other occasions, once for recurrence of carcinoma in a stricture following fulguration in a ninety-four year old patient and twice for bladder and prostatic rectal fistulas which were so extensive that they approached the nature of a cloaca. The results were equally good.

In addition to the necessity for continued observation of these patients, I should like to stress one point not previously mentioned. That is the extreme importance of excision of these lesions without trauma. Metastasis can occur at time of removal if the lesion is squeezed or clamped and in clinically malignant lesions it is wise to block the blood and lymph return before proceeding with difficult excisions.

Continued reexaminations of patients, even with obviously benign lesions, are important and the patient can be made to realize this without being frightened or made neurotic. In event of recurrence it is frequently not too late for more radical procedures aimed at really curing the patient.

The only point of disagreement with Dr. Binkley is in the use of x-ray and radium. My experience has been limited, admittedly due to my feeling that if I were the patient and had to

depend on radiation to eradicate cells growing beyond the reach of local excision or fulguration, I fear I should not sleep well. I need more convincing evidence that x-ray does anything to cells of colonic origin, especially when given via the usual portals.

In Dr. Swinton's thirty-five patients with adenopapillomatous lesions showing early malignancy which had not yet extended into the base of the lesion, he had no failures when the patients were treated with local excision. I have not been so fortunate. Within one year after local excision of such a lesion, an extrasigmoidal mass could be noted and I had to do a combined abdominoperineal excision at which time it was found that the only metastasis was about 4 to 6 inches away from the bowel which had apparently involved a lymph node.

W. J. Martin, Jr. (Louisville, Ky.): These have been two very comprehensive papers which I think will be a great addition to the literature. I think the discovery and eradication of these growths is more or less vital to the patient, because I think it is becoming generally accepted these things are definite precursors of cancer; and if we are careful in our examination and as we extend the number of examinations and the indications for the examinations, which has been brought out, we will find at least in some of these patients whom we can get to early that the eradication is not apt to be so radical.

In my series of cases I have found that approximately 7 per cent of the people that I examined have some type of papillary adenomatous growth in their bowel within reach of the sigmoidoscope. I do not believe that the gross recognition in determining whether or not these things are cancer or whether they are benign is very accurate. I think most of them that I find are above the finger, where you cannot palpate most of them. I think that the rectosigmoid growth is situated so that you cannot reach that high and do a good palpation on it in most cases. The gross appearance of them is very deceiving at times. I do not believe that too much stress should be placed on that. I think that microscopic diagnosis is the criterion. Of course, if there is a grossly ulcerated mass there in which all polypoid characteristics have been lost, that is a different story. These things usually occur singly, but with the help of the double contrast enema which is always used and always should be used if we find one, occasionally we find other growths higher up, which can be watched if one chooses to do that for a while, or radical surgery could be instituted at that time.

I think that in treating these things one has to be very cautious. It would be very nice if we could remove all of these by snare or fulguration and save people the risk of major surgery, but I think it should be emphasized to a group such as this that one should be very, very careful in choosing this type of treatment. The removal by snare and fulguration can be done by most anyone if he is cautious so that he will not run into bleeding; but I think if there is any one thing that we should carry away from here it is that the use of the fulguration machine and the snare should not be overemphasized.

Speaking of recurrences of these growths, sometimes I wonder if they are real, true recurrences. Why not a new polyp which has developed? Very frequently if, as I say, there is more than one and you remove the specimen surgically, you find small polyps adjacent or close to the original growth. It might be if one has done a local resection or has used the snare or fulgurating tip and removed a growth, it is perfectly possible that a small polyp might have been overlooked at the time and that the recurrence does not turn out to be a recurrence but an entirely new growth.

I believe that the age group has something to do with your choice. As one man brought out on prolapse of the rectum in an earlier paper, in these older people recurrence does not occur. I do not suppose their life expectancy was great enough to give them time for it to recur. I think in some of these individuals that life expectancy is short enough so that perhaps you might be a little more lenient and probably do a fulguration on some of those in the older group.

A. W. Martin Marino (Brooklyn, N. Y.): Tumors of the rectum are either benign or malignant. Benign tumors include fibromas, lipomas, angiomas, myomas and adenomas. The last named are the predominant type. Hyperplasia is not a form of neoplasm and should not be included in a discussion of neoplasms except from the standpoint of differential diagnosis. However, I might say at this point that any thickening or protrusion of the rectal mucosa demands careful investigation.

At times the term adenoma is loosely used as though some were benign and some malignant. When malignant transformation is proven, the lesion is no longer an adenoma but a carcinoma. Although adenomas are benign tumors, it should be kept in mind that they are premalignant lesions and therefore very important from the standpoint of early discovery as well as prompt and effective treatment. It is well to keep in mind, too, that glandular tumors of the large intestine may be multiple and for that reason the finding of an apparently isolated lesion in the rectum should make the examiner suspect the presence of other tumors, benign or malignant, higher up.

After I have become convinced that I am dealing with a solitary or with multiple papillary adenomas, all of which are within reach of the protoscope and amenable to local eradication, the treatment I employ is to remove the tumor or tumors *in toto* by means of the electrocoagulation snare with fulguration of the base. The removal of the lesion or lesions is accomplished in such a way as not to distort the specimen or specimens. To quote our pathologist at the Brooklyn Hospital, Dr. J. Arnold deVeer, "By all means have all tissue that is removed examined and the method of removal should be, if possible, the one that least interferes with histologic examination." I make it a rule to remove the tumor or tumors intact and not take a biopsy. Two things are accomplished by this procedure: first, the intact tumor permits serial microscopic study of the entire tumor and second, if the tumor proves to be benign and its removal has been properly accomplished and we are reasonably certain of its complete eradication, then the only thing that need concern us is the possibility of the development of a new lesion. For that reason we insist upon periodic check-ups of any patient who has been treated for an apparently isolated lesion.

I do not see any reason for removing a piece of a papillary adenoma for microscopic study when it is just as easy and certainly more satisfactory, so far as I am concerned, to remove the whole tumor and at the same time perhaps cure the patient all in one stage. In this connection I want to emphasize what Dr. Binkley said concerning the importance of estimating the malignant possibilities of the lesions under discussion by noting their clinical characteristics such as color, induration, ulceration and hemorrhagic tendencies. Such an estimation is a guide as to the form of treatment to be used. The final diagnosis, of course, is made by the pathologist.

In removing these small tumors through a proctoscope, general anesthesia is not needed because the cooperation of the patient is desired. Special instruments are essential: a good light, suction to remove smoke and fluid, snares and electrodes on long carriers, a reliable diathermy machine and a long forcep to recover the tumor after its detachment. Skill in the use of the proctoscope and experience in electrosurgery are prerequisites.

In certain fields of surgery, such as those dealing with the larynx and cervix uteri, an attempt has been made to recognize malignancy in epithelial membranes before there is frank invasion of the lamina propria or deeper layers. These lesions have been called "carcinoma *in situ*." In the case of rectal neoplasms, the criterion for recognition of malignancy should perhaps also be *not* actual invasion but early evidence of anaplasia prior to the invasion. In our hospital when the lesion is reported as malignant, a distinction is made in these relatively small tumors as to whether they are invasive or malignancies *in situ*. If it is a malignancy *in situ*, removal by electrocoagulation with fulguration of the base suffices in my experience. On the other hand, if the specimen exhibits invasion of the submucosa, I perform radical resection because that makes abdominal exploration for metastases possible and, furthermore, only by microscopic examination of all the coats of the bowel wall can I be certain of the extent of invasion of its coats.

In conclusion I would say that I divide rectal tumors into two classes: those that are benign and which can be cured by local eradication and those which are malignant. I divide the malignant tumors also into two classes: those that are malignant *in situ* and amenable to local eradication and those that require the more formidable forms of treatment. So far as I am concerned, prompt, adequate and radical surgery is our best weapon against rectal carcinoma, especially early carcinoma.

R. A. Scarborough (San Francisco, Calif.): I think very much we need a pathologist here for this symposium discussion. We are using terms more or less loosely. I think in some of the discussion we have missed the significance of Dr. Binkley's first paragraph, in which he

attempted to isolate one particular group of adenomatous tumors and classify them as papillary adenomas. All three discussants used the term "papillary adenoma" more or less generally, but this is a particular group of adenomas which give us the greatest concern. They are the most treacherous type of tumor which we have to deal with in the colon in making the decision as to what kind of a tumor it is.

Dr. Marino has classified his tumors as benign and malignant. I certainly think he needs a third classification of "or both," because in the majority of the papillary tumors which Dr. Binkley has described, biopsy will usually reveal the appearance of a benign tumor. In this particular type of tumor malignant change develops in the base of the tumor usually centrally located, hard to approach for biopsy, whereas in the ordinary adenomatous polyp which becomes malignant, degeneration occurs on the periphery and is subject to approach by superficial biopsy. Size in papillary adenomas has no direct relationship to the possibility of malignant degeneration in the base or the degree of malignancy present. We need not only a pathologist in helping us to decide about these tumors but I think we need a pathologic specialist. There are too many pathologists today in this country, certainly in my part of the country, who facilitate their diagnosis by calling every adenomatous polyp of the rectum or colon carcinoma grade I. I do not believe that helps us at all in our decision as to what to do for those patients.

We have to depend on our pathologist and a pathologist must be able to identify the changes not only of benign proliferation but of the type which Dr. Binkley mentioned as atypical changes, which also must be classified as benign, I believe, and not malignant. I would like to ask him to discuss that and see which group he places them in, because we believe that those should be classified as benign and treated as benign lesions. But when malignancy does develop in this particular type of papillary tumor, hidden perhaps somewhere in the base, the most radical treatment is in order.

Rudolph V. Gorsch (Flushing, N. Y.): I think that no matter how much experience you have had in proctoscopy or looking at these tumors, it is just impossible for you to tell whether some of these tumors are benign or whether they are malignant. Therefore, I think that one of the most important things to do is to try, as Dr. Marino says, to get the whole tumor. I know that everydody does not agree with that because the snare removal of a tumor, of course, (there is no question about it) is sometimes a little bit difficult and sometimes you are really apt to pinch a part of the bowel wall along with your tumor. But certainly I do not see how you can get anywhere at all unless you get the whole tumor out and examine the whole tumor, because, as has been brought out many times before, the periphery may be malignant or the base can be malignant. If you destroy the whole tumor, of course, you have no evidence at all.

Just a word about the technic of taking these tumors off with a snare. If you want to have a snare where you can push the loop in and out and adjust the snare wire over the top of the tumor, that facilitates the removal very, very easily. If you have a fixed wire there before you try to put it over the tumor, sometimes you are under a considerable amount of difficulty in getting the wire just where you want it on the pedicle. I never put forceps on the tumor at all because I think that is the way you are apt to pull the tumor out and snare part of the bowel wall along with it. The thing to do is to put a piece of gauze above the tumor or three or four pieces of cotton and the humor will fall right up there and you can pick it out later on.

The next important thing for you is to put your patient in a position so the pedicle of the tumor will hang down. Of course, you have to have proper apparatus; you have to have a proper table and all for that. Then you put your snare in there and you just put the wire around the pedicle and I think an important thing is to put the wire so you can turn the snare and you can actually see the pedicle move on the bowel wall. Then when you snare the tumor off, you have very little difficulty. You will have very little danger of including some of the bowel wall.

After I have done that, I invariably take a suction coagulation handle which I have devised and I (I do not say fulgurate) desiccate the base of the tumor. I desiccate a considerable area around the base of the tumor in order to destroy any lymphatics or possibly any tumor cell which is out around, possibly a centimeter around the tumor.

A distinction should be made in the terminology of the type of current which you are using.

Fulguration is a general term and I do not think proctologists should use it. You either use the Oudin current, the desiccating current or a coagulating current. There is a great deal of difference in the electrosurgical Oudin current and the coagulating current. I think we ought to know which current we are using and we ought to tell our audience which current we are using. Fulgurating is just a general term. With coagulating current you can get into a great deal more trouble because it penetrates the tissues more easily.

There is one other important point about this whole symposium, that is the follow-up. It has been my pleasure recently to present a series of patients treated by Dr. Yeomans many years ago and also to see patients treated by other proctologists around New York City. They come back to me and I get a history on them. In some instances they had had a polyp removed by Dr. Yeomans some twenty or twenty-five years ago. Then, in another case someone had a polyp removed six months ago. I said, "Did the doctor tell you to come back?"

"No."

Of course, obviously, part of the treatment is follow-up. If you take a tumor out, you do not know whether it is benign or malignant, no matter how much experience you have. The important thing for you to do is follow up the patients at least periodically every three months.

A. W. Martin Marino: Mr. Chairman, may I have a word?

President Ricketts: Yes.

Dr. Marino: The hardest decisions I have to make are in those adenomas which the pathologist reports malignancy. Then I go to the pathologist and we go over the situation together. I agree perfectly that a competent pathologist is a most necessary accessory in the performance of these operations. Fortunately, papillary adenomas make a small percentage of all adenomas and the question does not arise too frequently, but I have seen what have been reported as benign adenomas by competent pathologists. I have seen cases develop carcinomas on those sites. It may be that something went wrong.

By the same token, I have seen adenocarcinomas, papillary, small, (they talk of small lesions in this discussion) which have healed perfectly. The mucosa has been so restored that it would be almost impossible to find the location where that adenocarcinoma had been removed. In following these cases, a carcinoma developed behind. In other words, you can get a fine looking result on the outside; but, even though you have a biopsy, you do not know how much invasion of the various coats has taken place. You are never sure whether or not instead of curing that patient you have simply postponed the eventual diagnosis and perhaps hastened, if not caused, his death.

E. G. Martin (Detroit, Mich.): I simply want to warn against fulgurating or desiccating above the rectosigmoid. I do not think the Doctor intended to leave that impression, because you can very readily burn a hole into the peritoneal cavity.

I also would like to say that if we have a sessile tumor in the sigmoid, I believe it should be taken out. I want particularly to express my enjoyment and appreciation of Dr. Swinton's paper and Dr. Laird's discussion.

George E. Binkley (closing): I am not quite sure that I got over what I set out to do I was not discussing small tumors. I was discussing angiopapillomas, whether they were as big as the end of a pin or whether they were as big as two hands. Moreover, I was not discussing adenomas as we see them; I was discussing a brand, a breed of tumor which we ought to know something about as proctologists, if anybody should; I do not believe we do, I do not and I do not believe you boys do, either.

It is true there are two types of rectal tumors as far as one is malignant and one is benign. I am confining my remarks to the papillary adenoma which I think we ought to recognize and not mix up with everything else. If we do not recognize them, who will? It is possible to do it clinically if you will spend a little time and see enough of these tumors. There is no use saying it cannot be done just because it never has been done; because you cannot do it is not saying someone else cannot do it. We are going to go ahead and you have got to go ahead or stay static or go back; one usually goes back. It is possible clinically to recognize these tumors in a great many cases and they are real problems, as Dr. Scarborough brought out. He has had a little experience and he recognized them for years; he knows what we are talking about.

You take the benign and malignant lesions, yes, and the papillary adenomas, there are benign and malignant lesions but there was

scarcely anybody who recognized them, because I did not tell you much about those atypical changes. That is a pathologic or histologic job, but that is where they fall down. What good is your prognosis if your man or your pathologist does not recognize atypical changes? There are atypical changes, as I see them. Patients come to me with a diagnosis of carcinoma, grade anywhere from 1 to 4, and when I hand them over to Dr. Stewart at Memorial, he says, "There isn't a bit of malignant change in that site. There are atypical changes." It is still a non-malignant polyp. If you are going to take those out and treat them as malignancy, they may become malignant later; but I do not think that man wants a radical resection just because he happens to come and your man or you or somebody else makes a bum diagnosis and he loses his rectum. That is the point I am trying to get over here, that this type of disease has atypical changes and they are still polyps or non-malignant tumors while they have them, until they go on to the malignant group. You have to have a pathologist to recognize that, otherwise you will have 100 per cent five-year survivals no matter what you do with them.

I am not criticizing anybody. I am trying to get you gentlemen to see what I am driving at. Maybe I am all wrong; if I am, why then that is too bad. That is the point I am trying to get over. These things can be classified and the most mistakes are made by pathologists recognizing atypical changes and calling them some form of malignancy, 1, 2, 3 or 4. I can show you patients who have come with the slides and the diagnosis and I have made some enemies among my colleagues because I would not do a radical resection on a patient who had atypical change. They thought I was trying to get the patient away by telling him he did not need an operation.

Someone once took a rectum out of a patient with benign or atypical changes. Even some of the people who have the label of cancer on them have not even got atypical change. What most of you want is a good pathologist. I do not know what I would do without the one we have.

Another point is that Dr. Laird did not think much about radiation therapy. Well, this involves an old age group of patients as a rule; some of them are eighty-five, some are seventy-five, some seventy-eight. Do not think that you are going to cure all of those people with large papillary adenomas, some of them with atypical changes, some of them with carcinoma; but you can control them until they go out with a cardiac or some other complaint. If you are setting out to do radical resections in all the papillary adenomas, maybe you will get away with it; but I will turn some of mine over if you want to do radical sections. I do not believe I can do it. In that group of cases you can use your radiation to a good degree. You may not cure them, they may be symptom-free for a while and you may have to go back to your fulguration.

I am not going to be pulled into a discussion of treatment. That is too big a job for one day. This is a problem, I think, we should give a good deal of consideration to and it might be wise sometime to write it up and get two or three good pathologists to teach us something about it, because as clinicians we do not know any too much. We can certainly stand some straightening out and some good slides and it might be a benefit not only to ourselves, but also to the pathologists in our own hospitals.

Neil W. Swinton (closing): I appreciated Dr. Binkley's paper very much and I was particularly interested in it because I know exactly, I think, what he is talking about. There is very little in the literature. I see among the polyps we find in the clinic a large number of the type that he has described, which I have recognized as being a different entity in many ways from the true adenoma.

I would like to ask you one question. I have not been too impressed with the malignant potentialities of those turmos and yet I have had quite a number that have recurred locally over a period of years. I have had several of those patients whom I have followed between five and ten years. They come in and see me about every six months or once a year, and I find a local recurrence. I burn it off and they are all right for another year. I would like to ask him if he has had a similar experience.

Two or three other points: The use of radium and x-ray, I think, is a personal matter. Our department of radiology does not approve of its use in gastrointestinal malignancy and we have had no experience with it.

The case Dr. Laird mentioned with the early metastasis, I am certain, is going to happen to

us one of these days if we do many of these, and I think it must always be considered.

I wanted to say a word about x-ray in the diagnosis of polyps in general; I did not do it in the previous discussion and it was not brought out. We have been very unhappy in our department of radiology in the number of tumors that we are picking up at an early stage. We know we are not finding a great number of them. A lot was said about contrast air enemas and I believe that you must realize that you are going to miss certain tumors both ways. With proper and complete colon investigation, I believe very strongly they should have both a barium enema and a double contrast. We do them at different times and with a complete repetition of preparation of the colon for each examination.

Another thing, if we find a polyp or polyps, we have learned that we still cannot depend entirely on our radiologist and we will wait three or four weeks and repeat the double contrast air enema; if the polyp is in the same place, then we operate. Many times the polyp has moved.

I wanted to say one other thing, in general on this whole polyp question. Last year in the United States there were 296,000 people who died of malignancy. The last figures I saw on a breakdown for cancer of the colon and rectum were for 1943, at which time there were around 8,000 people dying of cancer of the rectum and colon. We have demonstrated there is a premalignant lesion. What percentage of all the cancer it is, I do not know. I personally think it is at least half. I think that the members of this group, representing certainly in this country the men most interested in this field, have a mission. I think that if we could have everybody in the United States over thirty-five years of age proctoscoped, you would be simply astounded at what we would find.

We are conducting at the clinic on a small scale a sigmoidoscopic examination as a part of a routine physical examination in older patients. We are not able to do it on a wholesale basis. Those figures I will have in another year or two, but we are finding premalignant lesions in patients over forty or fifty years of age in at least 7 per cent of all the patients who go through our office. I know we are saving many patients from cancer.

I enjoyed the discussion very much, and I wish to apologize to Dr. Marino and Dr. Laird in that my paper was negligently written very late and I was not able to send a copy to them in advance.

EXTRARECTAL AND EXTRASIGMOIDAL MASSES*

PROCTOSIGMOIDOSCOPIC INTERPRETATION AND EVALUATION

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MOST or many of us who practice in the field of proctology are engaged in the teaching of proctologic fundamentals to undergraduate and graduate students. It is a gratifying experience and while time-consuming is not without its rewards. For a number of years I have stated among other dogmatic assertions that no specialty in medicine can offer a higher percentage of accurate diagnoses than proctology. As we know all too well, the patient's history is often inaccurate and misleading. Thus, the carcinoma patient denies bleeding; constipation to one patient is diarrhea to another. The history rarely differentiates the disease but it should always direct attention to the need for a systematic, careful study of the anus, rectum and colon. With instruments of precision including an experienced index finger, the sigmoidoscope and the roentgen ray, the margin of error should be slim indeed. This is what we teach our students; it is of this that we talk when addressing county and other local medical groups.

During recent years it has become more and more apparent to me that there are certain intangibles in proctology. Most of us have been content with the common phrase "extrarectal or extrasigmoidal mass." Following our examination, which does not demonstrate but makes obvious the presence of such a mass, we submit a written report describing the location of such an impinging mass, its degree of fixation, its consistency, the presence or absence of tenderness and other rather nebulous conclusions. Rarely do we offer a suggestion as to its exact nature. This is probably a correct attitude because usually we do not know.

In reviewing my own records over a twelve-year-period I found to my great surprise that the diagnosis of extrarectal or extrasigmoidal mass or swelling had been entered into the cross files on 313 different patients. Similarly, I was delighted to find that an absolute diagnosis was ultimately established in 284 of these persons. It is thus possible to analyze such a group of case histories and to check the proctosigmoidoscopic findings and the tentative impression gained against the final definitive diagnosis.

A characteristic case history will outline the problem as it often presents itself:

CASE REPORT

Mrs. L. L., aged thirty-nine years, was referred to me in May, 1946 and brought to my office by her brother who is a physician. Briefly, her history of three months' duration was one of short episodes of diarrhea lasting a day or two. There was cramping in the lower abdomen. Twelve days prior to my seeing her, there was a sudden desire to defecate and she passed a small amount of bright red blood. She mentioned a sense of rectal pressure. There had been no weight loss. The cramps and diarrhea were not related to the menstrual periods. There were no other complaints. Proctosigmoidoscopic examination was attempted and I quote from my report. "On digital examination there is a very hard nodular mass felt in the cul-de-sac. It feels extra rectal. Sigmoidoscope passed 11 cm. At this point the mucosa is edematous and thrown into folds. The bowel is sharply angulated and the mass appears to be anterior to the upper rectum. No type of sigmoidoscope could be passed beyond this level. No blood streaks were seen. No intrinsic rectal lesion noted other than small internal hemorrhoids." It was my impression that the palpated lesion was malignant but its

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exact nature and origin was in doubt. Roentgenographic examination of the colon was attempted and the report was that there was a point of blockage of the upper rectum beyond which barium would not pass. (In passing it might be mentioned that the roentgenologist thought it indeed strange that I could not visualize such a low lying lesion.)

Surgical and gynecologic consultants were called upon and the final conclusion was that a pelvic malignancy existed and that exploratory laparotomy was justified. As a result of such pooling of our meagre knowledge, the patient was thoroughly prepared preoperatively so that surgery of any magnitude could be done if necessary. At operation a carcinoma of the mid-sigmoid was found which had become fixed to the lower part of the uterine fundus. Combined abdominoperineal resection including hysterectomy was done and to date the patient is quite well.

This case illustrates and perhaps exaggerates some of the difficulties encountered. In most instances the problem is much easier.

As I began to assemble these data a thorough review of all previously published material seemed to indicate that to the best of my knowledge only one prior effort has been made to classify the various extrarectal and extrasigmoidal masses and to determine their causation. This was a paper published in the Journal of the American Medical Association of July 19, 1944 by Drs. Buie, Jackman and Vickers. It was an excellent and concise presentation based upon 254 patients with malignant tumors of the rectovesical and recto-uterine spaces as had been noted by the examining proctologists of the Mayo Clinic.

An extrarectal mass may feel malignant but prove inflammatory and an occasional extra lower bowel mass may on palpation suggest inflammatory tissue and prove to be malignant. It seemed advisable, therefore, in this review to include all extrarectal and extrasigmoidal masses when on palpation or on proctosigmoidoscopic examination the exact nature of the mass could not be determined with accuracy.

What brings such a patient to the proctologist? Curiously, the symptoms were referable to the lower colon or rectum in 61 per cent of the total patients. In many instances the mass was felt by the patient's own physician and to his credit he had believed that proctoscopic examination was indicated. A few were referred for proctoscopic examination as a preliminary to roentgenographic studies of the gastrointestinal tract.

Analysis of the total (313) patients with extrarectal and extrasigmoidal masses revealed the following:

Table 1

TYPE AND ORIGIN PROVEN

Method of Proof:	
1. Biopsy	6
2. Roentgenograms	77
3. Gynecologic study	57
4. Urologic study	20
5. Laparotomy	51
6. Rectal operation	55
7. Autopsy	18
Total Cases with confirmed diagnosis	284
Total Cases with unconfirmed diagnosis	29

In twenty-two instances the failure of confirmation of diagnosis was refusal on the part of the patient to submit to further investigation. In seven fatal cases autopsy was not obtained.

It will be noted that biopsy as a means of obtaining microscopic proof of the diagnosis was rarely employed. It is with the greatest hesitancy that I offer to puncture an intact mucosa to obtain a microscopic section. Certainly I would never do so if I knew the mass was above the peritoneal reflection. Other and safer means exist to confirm one's suspicions. Since the term "rectal operation" in the above table would appear to be merely an extensive biopsy, I would hasten to add that in the series studied I have, perhaps unwisely, included all perirectal abscesses at or above the level of the levator ani muscles.

A detailed discussion of each of the above listed diseases is neither practical nor possible. However, a few general statements seem advisable: When the finger or the end of the sigmoidoscope encounters resistance and pressure causes intense pain, we are in

all likelihood dealing with an inflammatory rather than a neoplastic entity. Such subjective evidence is by no means absolute but it is highly suggestive. The duration of symptoms, if any, must be carefully analyzed. Thus, the patient who complains of

TABLE II
CONFIRMED DIAGNOSIS AND ORIGIN OF LESION IN 284 PATIENTS

Diagnosis	Total Cases
I. Perirectal or pelvic abscess—levator level or higher	47
II. Presacral cysts and dermoids	4
III. Pelvic disease (female)	77
(a) Leiomyomatous uterii and displacement	21
(b) Tubo-ovarian inflammation or abscess	29
(c) Endometriosis	17
(1) Pelvic organs	9
(2) Rectovaginal septum	5
(3) Sigmoid	3
(d) Ovarian cystic disease	4
(e) Carcinoma uterus, ovaries	5
(f) Carcinoma bladder	1
IV. Genitourinary tract (male)	20
(a) Seminal vesiculitis	2
(b) Carcinoma prostate	15
(c) Carcinoma bladder	2
(d) Prostatic abscess	1
V. Diverticulitis	74
VI. Rectal shelf implants (Blumer)	23
VII. Retroperitoneal sarcoma, carcinomatosis	11
VIII. Perirectal tumors	8
(a) Tumors of ischio anal fossa	5
(b) Perirectal carcinoid	3
(c) Squamous epithelioma in deep fistula tract	1
IX. Carcinoma sigmoid	20

pain deep in the pelvis of only a few days or weeks duration is again likely to be found harboring an inflammatory process. This might be a smouldering pelvirectal abscess, gynecologic or urinary tract infection and of course subacute, acute or ruptured diverticula of the sigmoid.

A hard, fixed, nodular, non-tender mass palpated through the anterior wall of the mid-rectum will rarely prove to be anything except carcinoma or malignant implants. The original lesion must then be sought. In this review the stomach seemed to be the primary focus in more than one-half of the patients.

It is known that an ulcerated inflamed diverticulum of the sigmoid will produce not only the symptoms of constipation or diarrhea or both, with increased gas and left lower abdominal pain, but may produce bleeding as well. It is my belief, however, that bleeding caused by diverticulitis is comparatively rare and to me this presence or absence of bleeding is the outstanding differentiating feature between sigmoidal carcinoma and diverticulitis.

Lesions of the genitourinary tract in the female which are producing low colonic symptoms are rarely of short duration. The past history is often illuminating. Periodicity of symptoms, especially if related to the menstrual periods is significant. Such a relationship in women with endometriosis is well known.

Reexamination of Table II should quickly convince the reader that establishing the ultimate correct diagnosis is rarely a "one man job." Help is needed and occasionally multiple consultations are required. The proctologist may speculate as to the underlying pathologic lesion but his conjectures should not cause undue delay.

If a shadow of a doubt exists as to the actual diagnosis (and such doubt will exist more times than not), then let him call upon his colleagues the roentgenologist, the gynecologist or the urologist. By such means ill advised or hasty laparotomy performed upon a poorly prepared patient will usually be avoided.

In mentioning the paucity of previously written reports, I would hasten to add that many splendid studies have been published each dealing with one of the single manifestations of extrabowel lesions. Thus, among others are the papers by Blumer concerning the rectal shelf implants; Masson, Cullen and many others on perirectal and perisigmoidal findings in endometriosis. Especially would I recall to your attention the excellent presentation by Jackman in 1942 on the proctosigmoidoscopic findings in diverticular disease of the sigmoid.

COMMENT

From this brief discussion of some of the features in diseased conditions producing extrarectal and extrasigmoidal masses it

can be seen that in many instances certain although perhaps vague characteristics are present. If critically analyzed, an attempt at differential diagnosis is justifiable. Certainly such an attempt is an interesting speculation.

One may properly ask: "Why should the proctologist worry as to the pathological nature of a lesion which he cannot see?" My answer, if one is needed, is that the proctologist in every possible way must lend his help and knowledge to the referring physician and for the benefit of the patient. The proctologist need not act as gynecologic, urologic or roentgenologic consultant. Others render such service but he should as a result of his examination be able to sit down with the patient and the referring physician to discuss the possibilities, the method of approach, the need for further consultation and perhaps touch on prognosis. Such a consulting proctologist will then have rendered a far greater service than merely to have written as his report "Extrarectal or extrasigmoidal mass: type unknown."

A CARBUNCLE is simply an extensive connected furunculosis. The Staphylococcus is the most frequently found organism. This lesion is quite uncommon in the perianal skin.

The brief excerpts in this issue have been taken from "Ambulatory Proctology" by Alfred J. Cantor (Paul B. Hoeber, Inc.)

PRIMARY RESECTION OF THE COLON AND RECTUM WITH PARTICULAR REFERENCE TO CANCER AND ULCERATIVE COLITIS*

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THE surgeon's primary concern with the problem of visceral malignancy up until quite recently has been with operative mortality. Perusal of the literature confirms this suggestion. The improvement which has come about in surgery generally within the last few years definitely suggests that the surgeon, now having improved considerably his record with reference to operative mortality, has begun to focus his attention more earnestly upon the problem of ultimate cure of cancer.

The perineal operation for rectal cancer and posterior excision of the rectum preceded by colostomy are reserved largely for the occasional patient for whom the conventional, abdominoperineal operation does not seem well suited. In most clinics professing an interest in the problem of rectal cancer the abdominoperineal operation has become the procedure of choice. In this clinic during the past five years, attempts have been made to ascertain whether an abdominal dissection directed at removal of the lesion and the lymph node drainage area, accompanied by reestablishment of intestinal continuity, is a satisfactory operation for rectal and rectosigmoidal cancers.

That such an operation can be done with an operative mortality not out of line with that of the abdominoperineal operation is apparent in our own experience; that the operation is followed by satisfactory continence in most patients, especially when the end-to-end suture method has been employed, also has been established; that this more conservative operation cannot have as high an ultimate cure rate for rectal cancer as the abdominoperineal operation is also apparent in that omission of the perineal portion of the operation, particularly salvage of the levators, constitutes a less radical operation for rectal cancer.

From our own experience it is yet too early to give an opinion in the matter of how much less efficient the more conservative operation is than the standard abdominoperineal operation. That is the particular point still to be clarified. Already in 1944, after two years' experience with the operation in which sphincteric function was preserved, it was apparent that the operation was unsuited for dealing with low lying lesions in juxtaposition to the levators,[14] as well as for large, bulky, fixed cancers presenting lymph node involvement.

Accumulated experience from a number of sources suggests definitely that no surgeon employing even the most radical operation can lay claim to any important achievement in dealing with rectal cancers exhibiting lymph node involvement which fall into Dukes' Group C Classification.[3,4] Earlier detection and earlier operation for rectal cancer are without question the items which must be looked to, in order that a more hopeful prospect will come about in the field of rectal cancer.

CANCER OF THE COLON

In cancer of the colon it is possible to do a one-stage excision with removal of the

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lymphatic drainage area, preserving intestinal continuity in all operable cases. The Bloch-Paul-Mikulicz operation is an incomplete operation for colic cancer, and should be discarded by all experienced visceral surgeons. No surgeon should be found advocating general adoption of the abdominoperineal operation for rectal cancer, at the same time employing the exteriorization operation for cancer of the colon. Such practice smacks of inconsistency and suggests that the surgeon's mastery of the abdominoperineal operation has superseded his accomplishment in the one-stage end-to-end anastomosis for colic cancer.

Our associate, Dr. David State,[10] has advocated an extension of the abdominoperineal operation for those patients who exhibit lymph node involvement in the upper end of the inferior mesenteric artery pedicle; in such patients, he excises the entire left colon, together with the mesentery, establishing a colostomy in the transverse colon. The latitude for this extension of removal of the lymphatic drainage area is even wider in anastomotic operations. When numerous and enlarged lymph nodes are found in juxtaposition to a colic lesion, it has been routine practice for some years in this clinic to remove a considerably larger segment of the colon, thus enlarging the opportunity of getting wide of the lesion. Especially in lesions of the left colon is such a practice desirable. Having observed instances of recurrence about the ureter and the aorta after incomplete excision of the lymphatic drainage area in cancers of the descending colon, excision of that segment together with the splenic and sigmoid flexures, establishing continuity by anastomosing the transverse to the terminal pelvic colon has become quite routine practice in this clinic for such lesions. Moreover, in a few instances, because of the finding of enlarged lymph nodes in the mesentery of even more proximal reaches of the colon, the greater portion of the colon has been excised, anastomosing the ileum to the terminal pelvic colon. In some instances the lymph nodes have been reported by the pathologist as benign. Yet, this circumstance is less embarrassing to both patient and surgeon than is incomplete removal of the lymph nodes involved by the cancer process. Surgeons must now and then have occasion to entertain some misgivings over such negative reports of lymph node involvement. Obviously the difficulty of recognizing a few cancer cells in a lymph node must be great. The biologic test constituted by the elapse of time, to be certain, is a sure test to determine whether the pathologist's observation was correct but it is not a risk to be run with the interests of the patient in mind. Excision of an increased length of colon does not change the character of the surgical problem particularly and if one were to adopt this policy uniformly, the surgeon would undoubtedly be rewarded sufficiently often by the finding of unsuspected polyps as well as an occasional additional undetected cancer to justify this extension of effort.

In malignancies of the right colon the sentinel node at the inferior border of the pancreas alongside the mesenteric vessels is to be sought out and removed. In cancers of the transverse colon, the splenic and hepatic flexures should be unhinged from their phrenocolic attachments, thus permitting removal of a wider segment of the mesentery, insuring at the same time adequate mobility of bowel to effect an end-to-end anastomosis without tension. Among the patients included in this study are three patients in whom it was necessary to anastomose the proximal third of the transverse colon to the rectum because of extensive lymph node involvement in the inferior mesenteric artery lymphatic pedicle. Inasmuch as sacrifice of a good portion of the sigmoid colon voids the possibility of restoration of continuity between rectum and descending colon, the only alternative is mobilization of the transverse colon for the anastomosis. If the marginal vessels are well developed, the mid-colic artery may even be divided near

its origin and when the hepatic flexure and ascending colon are freed up, there is ample length of gut for an anastomosis to the mid-rectum.

To those surgeons who are committed to employment of the open anastomosis, the antibiotics such as the sulfonamides employed as intestinal antiseptics or streptomycin are a great boon. The experience of this clinic with primary resection, employing the closed anastomosis in the preintestinal antiseptic era (1941 to 1943) strongly suggests that employment of antibiotics is not a *sine qua non* of a successful operation.[12] In that two year interval sixty-one consecutive colic resections were done including the rectosigmoid area with one death, a hospital mortality of 1.6 per cent which is an accomplishment we have been unable to duplicate since, even with preoperative administration of intestinal antibiotics to occasional patients.

In every operative series of some size, when one is dealing with cancer, there enters always the item of unavoidable deaths; that is, losses in the postoperative phase from coronary and cerebral artery thrombosis and similar causes. The time may come when even death from pulmonary embolism will be looked upon as an avoidable cause of death. In the 1941 to 1943 experience there were no unavoidable deaths; in the 1943 to 1945 period, during which seventy-eight colic resections were done, there were six hospital deaths, a mortality of 7.6 per cent; three of the deaths, however, were owing to unavoidable cause. In other words, in the first series we used up a lot of surgical luck in having no unavoidable deaths. The score was evened up by the law of averages in the second series, giving us an overall hospital mortality of 5 per cent for both series.[13]

THE PRESENT STUDY

It is the writers' intent in this effort to indicate what the experience of this clinic has been with anastomotic operations for rectal and rectosigmoidal lesions. It is still too early to assess fully the accomplishment of the low anastomosis in dealing with primary rectal malignancies. Yet, enough experience has been accumulated to suggest what the outlook may be for the dual objective of curing the cancer and, at the same time, salvaging sphincteric function.

A serious interest in the problem of the low anastomosis with preservation of sphincteric function in this clinic dates back to 1942. It is the experience of those years which will be reviewed herein. Essential data concerning the patients and the operations are to be found in the tables. We have divided somewhat arbitrarily the surgical problem into two categories: (1) Those lesions situated within 13 cm. from the anus are regarded as being present in the true rectum; (2) those lesions beyond 13 cm. and not more than 20 cm. from the anus are considered as being in the rectosigmoid area. The term rectosigmoid is also a rather arbitrary and not strictly an anatomic designation. However, prior to the BNA reclassification of the constituent parts of the pelvic colon and rectum, the colon pelvinum, that segment of the colon between the distal end of the sigmoid flexure and the present arbitrary upper limits of the rectum (the third sacral vertebra) was included in the rectum. Surgeons, generally, employ the appellation rectosigmoid to this segment of colon.

In others words, in this study the operations have been divided into a high and low group. No lesions situated beyond 20 cm. from the anus have been included in this study. The majority of the patients had primary carcinomatous lesions of either the rectum or the rectosigmoid. In addition, there is a miscellaneous group constituted by a variety of conditions including primary lesions of the rectum which on microscopic study proved to be benign, extrinsic tumors involving the rectum and a few instances of diverticulitis. There is also a third group constituted by patients with ulcerative colitis for whom primary intestinal resection and anastomosis was done.

In this latter group, inasmuch as it was considered desirable to include all the patients having primary resection for ulcerative colitis, there are one or two included in whom the lower level of the lesion was a little higher than the arbitrary 20 cm level from the anus.

OPERATIVE TECHNIC EMPLOYED

The majority of the patients were operated upon employing the end-to-end suture technic. In the main, it can be said that one can make a satisfactory anastomosis at a level lower than it is safe to cure cancer. Because of the depth of the wound, a single row of sutures must suffice for the anastomosis. Interestingly enough, the results have been so satisfactory that we have come to employ a single row of interrupted silk sutures (0000) as the standard manner in which to effect an anastomosis anywhere in the gastrointestinal canal. We employ the Lembert type of stitch, spacing the sutures approximately 3 mm. apart. This somewhat close placement of the stitches makes for a rather large number of sutures, approximately forty sutures in the ordinary anastomosis. A large and patulous stoma follows this scheme of anastomosis quite uniformly.

The low anastomosis has been made by suture from within the abdomen as low as 3.5 cm. from the anal orifice with primary healing without formation of a sinus or fistula. The number of instances in which an anastomosis has been effected at as low a level as 5 cm. from the anus without temporary fistula formation, however, is few. Moreover, inasmuch as our own experience suggests quite definitely that even in the early lesion the very low anastomosis is contraindicated because of the likelihood of incomplete removal, the necessity for performing anastomosis at levels below 5 cm. is not frequent. The abdomino-anal pull-through operation has a limited indication. Inasmuch as healing is slow with this method, we have come to employ complemental colostomy in all such instances. Complete fecal deviation can be achieved by a single loop colostomy.[14] Then, when the segment of the pelvic colon pulled through the anus has healed securely, the colostomy can be closed. Healing in the direct suture anastomosis is ordinarily rapid, permitting dismissal of patients from the hospital usually within a week of performance of the operation. In patients in the upper group of rectal lesions (14 to 20 cm.) dismissal from the hospital five to six days after operation is the rule, just as in colic resection in the more proximal reaches of the colon barring complication. The operation without complication is the surgeon's first objective. It is synonymous with low mortality as well as low morbidity. Details of the technic in the performance of these operations have been described elsewhere and will not be repeated here.

CASE GROUPING

From Table 1, it is to be noted that during the years 1945 and 1946 at the University Hospitals 78 per cent of patients admitted to the surgical wards for treatment of cancer of the rectum or rectosigmoid underwent some type of radical operative procedure. In 56.4 per cent of instances the abdominoperineal operation was done and in 43.6 per cent the more conservative procedure of excision of the cancer with sphincter preservation was done.

Among the eighty-seven excisions of the rectum or rectosigmoid with sphincter preservation reported herein, there were sixty-three (72.4 per cent) so-called curative operations; the remaining twenty-four (27.6 per cent) were palliative excisions with restoration of bowel continuity. In this study the following criteria have been employed to characterize the palliative resection: (1) hepatic metastases; (2) peri-aortic lymph nodes or evidences of peritoneal metastases other than in the inferior mesenteric lymphatic pedicle; and (3) indurated lesions presenting high grade fixation to adjacent structures. The majority of these palliative resections fell

into the first category, i.e., patients with demonstrable hepatic metastases.

Table 1 suggests, in the main, that the pathologic grading or division of cases essentially employing Dukes' classification is that essentially reported in larger series

TABLE I
RESECTABILITY RATE FOR THE YEARS OF 1945 AND 1946 FOR PRIMARY CARCINOMA OF THE RECTUM AND LOW PELVIC COLON

	No. Cases	Resectable	Nonresectable
Jan. 1, 1945 to Dec. 31, 1945...	41	34	7
Jan. 1, 1946 to Dec. 31, 1946...	59	44	15
Total Jan. 1, 1945 to Dec. 31, 1946.....	100	78	22

	No. Cases	Per Cent of Resectable Cases
Abdominoperineal operations... (Both years)	44	56.4
Anastomotic operations.....	34	43.6

in which the abdominoperineal operation has been carried out. In a study of the St. Marks Hospital material, Gabriel[5] reported the following division of their cases: Dukes' group A, 15 per cent; group B, 36 per cent and group C, 49 per cent. In this present series of anastomotic procedures, resection was undertaken as a palliative operation in twenty-four patients or 27.6 per cent of the eighty-seven patients in the group. Of the eighty-seven patients subjected to operation (Table 1) 52 per cent fell into Dukes' groups A and B and 48 per cent into Dukes' group C. However, the division between the A and B cases was somewhat different in the present series, 26 per cent in each group, as contrasted with 15 per cent for Group A and 35 per cent for Group B as reported in the study by Gabriel in 1936.

However, amongst the sixty-three curative operations undertaken in this series, the division of patients was as follows: Dukes' group A, twenty-three patients (36.5 per cent); group B, eleven patients (27 per cent) and in group C, twenty-three patients (36.5 per cent). In other words, the case alignment in the curative group of

TABLE II
CANCER OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS
January, 1942 to June, 1947

Pathologic Classification of All Cases

Dukes' Group	No. Cases	Per Cent
A	23	26
B	23	26
C	41	48
	87	100

Pathologic Classification Curative Cases

Dukes' Group	No. Cases	Per Cent
A	23	36.5
B	17	27.0
C	23	36.5
	63	100.0

operations suggests definitely some case selection.

SUMMARY OF RESULTS

It is obviously too early to attempt to assess in a final manner the results of treatment of the more conservative operation in the cure of cancer of the rectum and rectosigmoid. Our experience with the procedures is summarized in Tables II to XI inclusive. This analysis is recorded in some detail in the hope that a study of the tables may prove helpful to others in assessing the worth of anastomotic operations in dealing with cancer of the rectum. Briefly summarized, these data suggest: (1) Restitution of intestinal continuity may be reestablished as a one-stage operative procedure after excision of the terminal pelvic colon and upper rectum with a hospital mortality that is in keeping with

that attending performance of the abdominoperineal operation. (Tables III and IV.)

TABLE III
DIVISION OF CASES AND OPERATIVE MORTALITY RESECTION WITH PRESERVATION OF THE SPHINCTERS FOR ALL LESIONS OF THE TRUE RECTUM AND LOW PELVIC COLON
January, 1942 to June, 1947

Procedure and Indication	No. Cases	No. Deaths	Per Cent Mortality
A. Resection and Primary Anastomosis			
1. Primary cancer of the true rectum (0–13 cm.)	51	3	5.88
2. Primary cancer of low pelvic colon (14–20 cm.)	20	1	5.00
3. Secondary malignant invasion			
a. Carcinoma of the ovary	2	0	0.00
b. Carcinoma of the cecum	2	0	0.00
4. Benign polyps	5	0	0.00
5. Diverticulitis	4	0	0.00
6. Benign post-irradiative stricture	1	0	0.00
7. Endometriosis	2	0	0.00
8. Non-specific ulcer of the rectum	1	0	0.00
9. Chronic ulcerative colitis	11	0	0.00
10. Tuberculous stricture of the rectum	1	0	0.00
11. Restoration of continuity after colostomy and inversion of rectal stump	1	0	0.00
Total	101	4	3.96
B. Resection and Pull-through of the Colon			
1. Primary cancer of the rectum (0–13 cm.)	16	1	6.25
2. Retroperitoneal lymphoblastoma	1	0	0.00
3. Perirectal abscess and fistula	1	0	0.00
Total	18	1	5.55
C. Resection and Pull-through of Ileum for Chronic Ulcerative Colitis	2	0	0.00
Total All Cases	121	5	4.13

(2) Functional sphincteric control is *complete* after the suture operation.

(3) Accompanying the abdomino-anal pull-through procedure good sphincteric control is the rule although 100 per cent recovery of function is not consistently

TABLE IV
CARCINOMA OF THE TRUE RECTUM AND LOW PELVIC COLON HOSPITAL MORTALITY IN PRIMARY RESECTION AND PRESERVATION OF THE SPHINCTERS

Type of Procedure	No. Cases	No. Died	Per Cent Mortality
A. Carcinoma of the true rectum (0–13 cm.)			
1. Resection and primary anastomosis	51	3	5.88
2. Resection and Hochenegg pull-through	15	1	6.65
3. Babcock-Bacon procedure.	1	0	0.00
Total	67	4	5.97
B. Carcinoma of the low pelvic colon (14–20 cm.)	20	1	5.00
Total	20	1	5.00
Total All Cases	87	5	3.76
Causes of hospital mortality			
1. Pulmonary embolus	2		
2. Coronary thrombosis	2		
3. Uremia	1		
Total	5		

obtained; moreover, the healing phase is longer and it is wise to make a complemental diversionary colostomy to afford rest to the pelvic colon pulled through the remaining distal sphincteric segment. Maintenance of normal continence in the suture operations is owing to preservation of the internal sphincter. Even in suture anastomosis made at 4 or 5 cm. from the anus, continence in these patients has been invariably good. Any operation[1] which eliminates the internal sphincter excludes the possibility of satisfactory continence. Even in the pull-through operation as done in this clinic, in which the rectal mucosa near the anorectal line is removed, (Whitehead maneuver), some injury to the

Table V

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS AND RESECTION (BOTH GROUPS) WITH ABDOMINO-ANAL PULL-THROUGH

January, 1942 to June, 1947

No. cases 87
Hospital mortality 5
No. follow-up 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	13	3	10	6	60.0	1	10.0	3	30.0	1	10.0
1943	13	2	11	8	72.7	2	18.2	1	9.10	1	9.10
1944	18	8	10	5	50.0	4	40.0	1	10.0	2	20.0
1945	14	2	12	8	66.7	4	33.3	0	0.00	2	16.6
1946	19	3	16	13	81.2	1	6.30	2	12.4	1	6.30
1947	5	1	4	4	100.0	0	0.00	0	0.00	0	0.00
Total	82	19 (23.2%)	63 (76.8%)	44	70.0	12	19.0	7	11.0	7	11.0

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

Table VI

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS FOR LESIONS 13 TO 20 CM. (UPPER GROUP)

January, 1942 to June, 1947

Total cases 20
Hospital mortality 1
No follow-up 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	3	1	2	1	50.0	0	0.00	1	50.0	0	0.00
1943	3	1	2	1	50.0	0	0.00	1	50.0	0	0.00
1944	4	3	1	1	100.0	0	0.00	0	0.0	0	0.00
1945	4	1	3	2	66.7	1	33.3	0	0.00	0	0.00
1946	4	1	3	2	66.7	0	0.00	1	33.3	0	0.00
1947	1	0	1	1	100.0	0	0.00	0	0.00	0	0.00
Total	19	7 (36.8%)	12 (63.2%)	8	66.7	1	8.30	3	25.0	0	0.00

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of he rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the toriginal lesion.

TABLE VII

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS AND RESECTION WITH ABDOMINO-ANAL PULL-THROUGH (0 TO 13 CM.) (LOWER GROUP)

January, 1942 to June, 1947

No. cases 67
Hospital mortality 4
No follow-up 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	10	2	8	5	62.5	1	12.5	2	25.0	1	12.5
1943	10	1	9	7	78	2	22.2	0	....	1	11.1
1944	14	5	9	4	44.4	4	44.4	1	11.1	2	22.2
1945	10	1	9	6	66.7	3	33.3	0	0	2	22.2
1946	15	2	13	11	84.6	1	7.7	1	7.7	1	7.7
1947	4	1	3	3	100.0	0	0	0	0	0	0
Total	63	12 (19.0)	51 (81)	36	70.6	11	21.6	4	7.8	7	13.7

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

TABLE VIII

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION AND HOCHENEGG PULL-THROUGH FOR LESIONS (0 TO 13 CM.)* (LOWER GROUP)

January, 1942 to June, 1947

Total cases 16
Hospital mortality 1
No follow-up 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	4	0	4	2	50.0	1	25.0	1	25.0	1	25.0
1943	5	0	5	4	80.0	1	20.0	0	0.00	1	20.0
1944	1	1	0								
1945	0	0	0								
1946	5	1	4	3	75.0	1	25.0	0	0.00	1	25.0
Total	15	2 (13.3%)	13 (86.7%)	9	69.2	3	23.1	1	7.70	3	23.1

* No abdomino-anal pull-through operations in the upper group (13–20 cm.).
[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

internal sphincter probably occurs, for the continence in this group of patients is by no means as complete as in the suture group. Moreover, as contrasted with the frequent loss of the sex function in males who have undergone abdominoperineal patients approximately the same promise of cure as does the more radical abdominoperineal operation. For lesions in this area presenting dubious or borderline operability the abdominoperineal operation is the procedure of choice.

TABLE IX

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS FOR LESIONS 0 TO 13 CM. (LOWER GROUP)

January, 1942 to June, 1947

Total cases 51
Hospital mortality 3
No follow-up 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	6	2	4	3	75.0	0	0.00	1	25.0	0	0.00
1943	5	1	4	3	75.0	1	25.0	0	0.00	0	0.00
1944	13	4	9	4	44.5	4	44.5	1	11.0	2	22.3
1945	10	1	9	6	66.7	3	33.3	0	0.00	2	22.3
1946	10	1	9	8	89.0	0	0.00	1	11.0	0	0.00
1947	4	1	3	3	100.0	0	0.00	0	0.00	0	0.00
Total	48	10 (20.9%)	38 (79.1%)	27	71.0	8	21.0	3	8.00	4	10.5

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

excision of the rectum, preservation of that function is the rule in the low anastomosis.

(4) In lesions of the rectosigmoid (upper segment 14 to 20 cm.) local recurrence has not followed the type of operation described herein. (Table VI.)

(5) The incidence of local recurrence following conservative procedures for lesions at 8 cm. or less from the anus is frequent enough to suggest that sphincter saving operations are contraindicated in all low-lying lesions; with abandonment of the conservative operations for low-lying lesions there will be less necessity for performance of the abdomino-anal pull-through operation.

(6) In the middle rectal segment or rectal ampulla above 8 cm. the conservative operation is a satisfactory operation for *suitable* cases and holds out to such

LOCAL RECURRENCE

The incidence of local recurrence in the curative groups is outlined in Table XI. Among these fifty-one patients operated upon, local recurrence has been observed in seven or 14 per cent of instances. Among these instances of local recurrence five or 70 per cent occurred in lesions at 8 cm. or less from the anus; two or 30 per cent occurred in the resections done for lesions between 9 and 13 cm. from the anus. One of these patients had a large colloid cancer which is notably difficult to cure;[7] the other had a large Dukes' group C lesion in which it became necessary to bring down the proximal third of the transverse colon for anastomosis with the mid-rectum because of the presence of large lymph nodes beyond the reaches of the conventional

excision of the inferior mesenteric artery lymphatic pedicle. Among the twenty-four patients upon whom palliative resection was undertaken local recurrence was observed three times; twice in the groups in which the lesions were 6 to 8 cm. from the anus and once in the group in which that lesion was 9 to 13 cm. This latter patient exhibited all three of the features which were designated above to characterize the palliative operation in this study.

Almost invariably, the local recurrence has not been *primarily* at the site of the anastomosis, but outside of it, extending into the bowel secondarily, a circumstance which suggests that it is the lateral invisible spread of cancer that the more conservative operation fails to deal with adequately. On this score, however, no operation is free from blame. David and Gilchrist who have been keen exponents of the necessity of liberal excision of the lymphatic drainage area in operations for cancer of the rectum, find on a study of their cases of abdominoperineal operation that the incidence of local recurrence amongst the five-year survivors was 22 per cent.[3] In other words, the abdominoperineal operation, the most radical of available curative operations, is also an incomplete operation for certain cases. That it may be for any lesion that extends beyond the longitudinal muscles of the rectal wall is apparent on careful scrutiny of any anatomic atlas which depicts the compartments of the pelvic fascia.

Local recurrence is an indictment of any operation and is synonymous with inadequate excision. It is to be noted (Table VI) in those instances in which the curative

TABLE X

CANCER OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS
Pathologic Classification and Survival Rate by Years
Curative Group

Year	Duke's Class	No. Cases	Exp. Ca.		Exp. Other		Living		Survival
			No.	Per Cent	No.	Per Cent	No.	Per Cent	
1942	A	4			1	25	3	75	5 yr.
	B	4	1	25	1	25	2	50	5 yr.
	C	2			1	50	1	50	5 yr.
1943	A	4					4	100	4 yr.
	B	4	1	25	1	25	2	40	4 yr.
	C	3	1	33			2	67	4 yr.
1944	A	5	1	20	1	20	3	60	3 yr.
	B	2					2	100	3 yr.
	C	3	3	100			0	0	3 yr.
1945	A	3	1	33			2	67	2 yr.
	B	1	1	100	..	0	0	0	2 yr.
	C	8	2	25			6	75	2 yr.
1946	A	4			2	50	2	50	1 yr.
	B	6	1	17			5	83	1 yr.
	C	6					6	100	1 yr.
1947	A	3					3	100	Less than 1 yr.
	B	0	..	..	..	..			
	C	1					1	100	
Total..	..	63	12	19	7	11	44	70	

operation was undertaken for lesions at 14 to 20 cm. from the anus, there were no local recurrences. This circumstance suggests rather definitely that for lesions in the rectosigmoid the conservative operation is probably just as effective for the cure of cancer as is the abdominoperineal operation.[15]

TABLE XI
CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS

Cases of Local Recurrence in the Curative Group

Distance in Cm. from Anus	No. Cases	No. Cases of Recurrence	Per Cent of Cases	Per Cent of Total Local Recurrence
0–5 cm. incl.	7	2	30	30
6–8 cm. incl.	12	3	25	40
9–13-cm. incl.	32	2	6.3	30
14–20 cm. incl.	12	0	0	0
Total	63	7	14	100

The very circumstance that recurrence is observed after the excision of a low-lying Dukes' Group A lesion outside of the site of anastomosis suggests that this or any other classification subdivides cases occasionally beyond what is justified by subsequent developments. In other words, a demonstrated Dukes' Group A lesion occasionally is shown by the elapse of time and growth of the lateral invisible spread to belong actually in Dukes' Group C. A closer correlation between the presence of venous invasion by the tumor and the Dukes' grouping is in order. That such venous invasion is rather frequent as well as ominous has been established.[2,9] Careful study to note the absence or presence of such venous invasion is important to assess thoroughly the possibility of cure in any lesion. Unfortunately, such a study was not made routinely in this group. It is certain that as one enlarges the indications for operation the number of patients with occult metastases will be larger.

UNSUSPECTED POLYPS IN A MORE PROXIMAL SEGMENT OF THE COLON

The frequency with which unsuspected polyps were encountered in this series is startling and suggests that if one were determined to leave no stone unturned to leave such precursors of colic cancer behind, he probably would be fully justified in excising the entire left colon, anastomosing the proximal third of the transverse colon to the rectum, a feasible operative procedure and one which was performed three times in this series of cases. As was stated previously for lesions in the descending colon, it has become regular practice in this clinic to excise the splenic and sigmoid flexures, anastomosing the transverse colon to the terminal pelvic colon. Certainly when one does the abdominoperineal operation for cancer in the lower and mid-rectal segments it is wise always to remove from above early in the operative procedure that segment of the sigmoid colon beyond the site chosen for colostomy down to the peritoneal reflection for purposes of inspection by the pathologist. Every now and then, the finding of a polyp or even a small flat independent cancer, undetected by sigmoidoscopic examination, may reward the surgeon for the observance of a caution which by some may be regarded as unnecessary. Such findings suggest the necessity for continuing the excision of segments proximally until no further lesions are encountered. And when one is contemplating anastomotic operations the proximal third of the transverse colon is a good stopping place, even when the anastomosis is to be made to the mid-rectum.

In this connection an interesting report of Mayo and Schlicke[6] of an autopsy study of polyps in the colon is of some importance. In patients coming to autopsy in whom the primary lesion was cancer of the colon or rectum, an additional polyp or polyps were found in 34.1 per cent of all patients. Moreover, an independent unsuspected cancer was found in 4.1 per cent

of instances. In patients dying of disease other than primary cancer of the colon or rectum, the incidence of polyps was 14 per cent. The incidence of polyps reported in the Mayo Schlicke study is considerably higher than is reported generally in barium and proctoscopic studies. In examinations in living patients, such unsuspected polyps are found ordinarily in 2 to 7 per cent of instances. Inasmuch as the polyp is a frequent precursor of colic or rectal cancer, it is certain that additional unsuspected polyps are more likely to be found in patients who come for the treatment of rectal or colic cancer. In any case it behooves the surgeon operating for malignancy of the colon or rectum to beware lest he overlook a precursor of a malignancy or an already existing independent cancer. If the Mayo and Schlicke observations are borne out in similar future studies, the surgeon who undertook to extirpate regularly the entire left colon, the area in which polyps are found most frequently, for lesions involving either the rectum, rectosigmoid or sigmoid colon, could probably justify his rashness by the finding of unsuspected potential malignancies or already existing independent cancers in sufficient numbers to justify the extra labor. Swinton and Haug[11] report upon a similar autopsy study of 1,843 patients. They observed an incidence of benign polyps in 7 per cent of these patients. In 42 per cent of the patients with polyps two or more benign lesions were present.

EARLIER RECOGNITION

Whether we deal with the point of entry or exit or intermediate conduits or reservoirs in between the portals of the alimentary tract, we all know how very important is early diagnosis for the successful management of cancer. Much as we lament the failure of patients to heed the warning of blood in the stool, pain or slight change in the bowel habit with reference to cancer in the lower reaches of the alimentary tract, yet an even more primary concern is that visceral cancer is a silent disease. The length of time involved in the transformation of normal epithelial cells from a normal mucous membrane into malignant cells is not known; how long it takes before invasive features cause the appearance of symptoms is undoubtedly a variable period, perhaps usually in excess of a year. Inasmuch as cancer of the alimentary canal including the colon and rectum is frequent and because cancer is an insidious and silent disease, we should take the pains to carry this instruction to the public; moreover, we should implement means of enlisting the public's concern and co-operation in thwarting the menace of latent cancer.

Whereas visceral cancer may occur at any age, the majority of cases of cancer of the colon and rectum are observed in patients beyond fifty years; in this series of rectal cancers, the extremes of age were thirty-one and eighty-six years; only 10 per cent of the patients were under forty-five years. In gastric cancer there appears to be a sharp rise of such cancers in the male at approximately fifty years of age. In colic and rectal cancers this rise in incidence appears to occur somewhat earlier. Until more specific means of detecting the presence of cancer are available, our only hope for more consistent early recognition of cancer is the routine examination of populations in which cancer may be developing by precise technics of examination. Such pilot cancer detection centers will, we believe, establish the validity of the thesis that silent cancer can be diagnosed. That many adults will have cancer is a foregone conclusion. It is merely a question of *who* will have it, in *what* organ and *when*.

We are concerned over making a more important impact upon the important problem of rectal and colic cancer than our present accomplishment suggests. It would be eminently fair to say here that the labor and the cost of operating a cancer detection clinic would be fully justified on the score of the digital and endoscopic findings attending examinations of the rectum and

lower sigmoid alone. Future discoveries may probably make the labors of such specialized routine examinations less necessary.

We must be realistic, however, and deal with situations as we find them. The surgery of malignant disease of the rectum and colon can be done with little risk. The only promise for more enduring results is that a larger number of patients come to operation when the lesions are silent and curable by surgery. When that time comes, the conservative operation which salvages sphincteric function will have an even more important rôle than it has now in the management of malignancies in the middle and upper segments of the rectum.

LOW ANASTOMOSIS FOR EXTRARECTAL MALIGNANCIES AND MISCELLANEOUS CONDITIONS

Some of the most difficult surgical problems were constituted by a miscellaneous group of patients necessitating colorectal resection, in whom restoration of intestinal continuity was re-established. Some of these fell in the upper group (14 to 20 cm.) and some in the lower (0 to 13 cm.). There were two patients with malignant tumors of the ovary involving the rectum and uterus or vagina. One of these patients when weighed directly following the excision of the enormous tumor was 15 pounds lighter despite liberal hydration and generous administration of blood during a long operative procedure. This tumor had been explored twice elsewhere and had been said to be inoperable. In addition to the rectal resection, a jejunal and bladder resection also were necessary in this individual. The colorectal anastomosis fell in the higher group. The other patient had undergone a previous right colectomy for cancer and a subsequent subtotal hysterectomy. In this instance the cancer of the ovary involved the rectum, cervix and upper third of the vagina. The colorectal anastomosis was made 5 cm. from the anus; the wound healed by first intention without sinus or fistula formation. It was looked upon as a palliative procedure but she has remained well for three years since operation with excellent sphincteric function. In one patient with a huge cancer of the cecum a fistulous communication existed with the rectosigmoid necessitating two simultaneous anastomoses. The lower colorectal anastomosis belonged to the upper group and the ileum was anastomosed end-to-end to the transverse colon. Table III lists the cases. An additional patient bears mention. She had tuberculous peritonitis and a colostomy had been performed in 1941 because of a stricture in the lower gut. At a subsequent operation an extensive tuberculous peritonitis of the pelvic colon was encountered. Considerable necrotic material in juxtaposition to the pelvic colon was curretted away, two simultaneous intestinal resections being carried out in the right and left colon. A stricture persisted at 10 cm. from the anus and ultimately a low anastomosis was carried out with subsequent closure of the colostomy. Inasmuch as the colostomy was of the Devine type, the only such colostomy ever made in our hospital, the closure had to be effected by excision of the colostomy segments and end-to-end anastomosis. Just as effective fecal deviation can be achieved by a single loop colostomy.[14]

PRIMARY RESECTION FOR CHRONIC ULCERATIVE COLITIS

There is a small group of patients with chronic ulcerative colitis for whom a primary resection can be done. Amongst the operations listed herein, there were thirteen such patients. The operations performed are listed in Table XII and the distribution of the disease is outlined in Table XIII. That such resections can be carried out in patients with segmental distribution of ulcerative colitis is understandable; a number of the patients with ulcerative colitis had that type of lesion. (Table XIII.) However, there were two patients in the group in whom the lesions of ulcerative colitis were present through-

out the colon and rectum, in which anastomosis of the ileum to the terminal pelvic colon, accompanied by simultaneous excision of the remainder of the colon, was followed by healing of the lesions in the rectum. Obviously, the condition of the mucosa of the distal rectal segment must be such that there is a possibility of recovery of its function after elimination of the greater portion of the disease. It had been suggested that the making of an anastomosis under such circumstances would probably lead to the upward migration of the disease from the remaining rectal segment into the lower reaches of the ileum. As a matter of fact, the reverse occurred. The disease in the rectal mucosa cleared up. In one instance this clearing process was slow as was indicated by the persistence of a tendency for the mucosa to bleed on proctoscopic examinations and by the continuance of diarrhea (four to six stools a day). In the remaining patient the improvement in general health was paralled by an early disappearance of proctoscopic evidence of the disease from the lower rectal segment.

TABLE XII
CHRONIC ULCERATIVE COLITIS
SURGICAL THERAPY WITH PRESERVATION FOR THE RECTUM OR A PORTION OF IT
January, 1942 to June, 1947

Surgical Procedure	No. Cases	Per Cent Mortality
A. Resection and Anastomosis		
1. Subtotal colectomy and ileocolostomy in terminal pelvic colon	4	0
2. Subtotal colectomy and ileoproctostomy	4	0
3. Partial colectomy and ileocolostomy left colon	1	0
4. Right hemicolectomy and ileotransverse colostomy	1	0
5. Subtotal colectomy and cecocolostomy in terminal pelvic colon*	1	0
Total	11	0
B. Total colectomy and pull-through of ileum	2	0
Total of All Cases	13	0

* This patient was operated upon in 1940.

This experience suggests that in suitable instances of ulcerative colitis, at an earlier stage in the disease, subtotal colectomy; with anastomosis of the ileum to the terminal pelvic colon or rectum, may lead to a disappearance of the residual lesions from the rectum.

TABLE XIII
CHRONIC ULCERATIVE COLITIS
Distribution of Diseased Areas

Site of Disease	No. Cases
1. Confined to rectum	1
2. Confined to colon	2
3. Colon and rectum	3
4. Colon and terminal ileum	6
5. Rectum, colon and terminal ileum	1
Total	13

One patient with a segmental type of lesions involving the colon as far distally as the sigmoid was quite febrile at the time of operation and had been so for weeks; subtotal colectomy was followed by immediate subsidence of fever and general improvement.

Two patients in the group had involvement of the entire colon with a stricture in the rectum at 10 cm. from the anus, with a normal mucosa in the distal rectal segment. Colectomy with anastomosis by suture to the distal rectal segment was carried out in these two patients with a very satisfactory result. Both patients are young women and despite the circumstance that the bowels move three or four times a day, they have excellent continence and bear this burden with no complaint.

In two patients presenting evidences of complete destruction of the large areas of the mucosa of both colon and rectum, a complete colectomy and partial rectectomy together with excision of the rectal mucosa from the distal segment was carried out, pulling the ileum through at the anus. The first of these operations was done on a young school boy five years ago, but the ileostomy has been closed only a few months. Despite quite satisfactory con-

tinence, the bowels move every two hours day and night because of the diarrheal character of the stool. Obviously, if this situation continues, the boy would be better off with an ileostomy bag.* In the other patient, the ileostomy has not yet been closed.†

In order to avoid impotence which quite often attends performance of the abdominoperineal operation for excision of the rectum in the male, colectomy and partial proctectomy with excision of the mucosa from the distal rectal segment has been carried out in one patient (not in this series) with simultaneous establishment of an ileostomy. The abdomino-anal pull-through operation for ulcerative colitis described above was proposed for this patient but he rejected it. This operation done more than five years ago was followed by a very satisfactory result with preservation of a normal sex function.

SUMMARY AND CONCLUSIONS

The experience of this clinic with restoration of intestinal continuity after excision of primary cancer of the rectosigmoid and rectum has been reviewed. The following conclusions seem warranted:

1. For all lesions in the rectosigmoid area 14 to 20 cm. from the anus, the conservative operation affords the patient as satisfactory a prospect of cure as does the abdominoperineal operation. In any case, no local recurrences have been observed after resections for cancers at this level.

2. For lesions in the lower rectal segment, at 8 cm. or less from the anus, the conservative operation is not a good operation for primary rectal cancer, primarily because it does not deal effectively with the invisible lateral spread of cancer as does the more radical abdominoperineal resection. In low-lying lesions a fairly high incidence of local recurrence stongly suggests that salvage of the rectal sphincters is accomplished at the risk of failing to cure the cancer.

3. For suitable lesions in the mid-rectal segment, above 8 cm. from the anus, the conservative operation would appear to be as satisfactory as the abdominoperineal; however, for fixed lesions, the abdominoperineal operation, undoubtedly the most radical operative procedure available, offers some advantage, the extent of which cannot yet be completely assessed.

4. The experience with the low anastomosis also is reviewed in a series of extrinsic rectal tumors as well as in a miscellany of other indications in which the lesion together with a portion of the rectum was excised with re-establishment of intestinal continuity.

5. The experience with primary restoration of intestinal continuity after excision of subtotal lengths of the colon and rectum for a small, select group of patients having chronic ulcerative colitis is recounted.

6. Finally, it may be said that satisfactory rectal continence attending these operations, particularly the suture methods of restoring continuity, is quite uniform with consistent preservation of a normal sex function. The importance of preservation of the internal sphincter for the maintenance of fecal continence cannot be overemphasized. A satisfactory anastomosis made by the suture method 3 to 4 cm. above the pectinate line assures complete continence.

* Since this was written, this patient has undergone vagotomy with considerable improvement. Vagotomy was done for two reasons: (1) to slow the gastric emptying time, (2) to attempt to abolish the gastro-ileac reflex.

† Since this paper was written, Drs. Ravitch and Gabiston of Baltimore have described carrying out such a procedure on the dog.[8] Their article is also well illustrated. The procedure was described briefly by one of us four years ago.[12]

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DISCUSSION

Thomas E. Jones (Cleveland, Ohio): I would like to say that unfortunately too many people in this country today think that the anastomotic operation is something new. Much has been written about it without reference to things in the past and the younger men at any rate think that this one-stage operation is a new operation. Well, I looked over my figures for the past twenty-seven years since 1920 and I am surprised at the number of one-stage end-to-end anastomoses that I made then and without complementary colostomy. Unfortunately, many of the people who are writing about this subject put complementary colostomy in fine print down below. I say that because people have come to my clinic and asked me if I do the primary resection. I said, "What is your definition of a primary resection?"

"Well, just cutting it out and sewing it together and dropping it back and that is the end of it."

Well, that is fine. I did it many, many years ago, but I changed to something that I believe is safer and now I do the great majority of operations by the exteriorization method. The reason that I did the end-to-end anastomosis in those days was this: I had seen many cases of local recurrence in the abdominal wall following a Mikulicz procedure. Therefore, I would not do one. I did the end-to-end anastomosis with or without tubes with the colostomy above.

Then I came across the Rankin modification of the Mikulicz which appealed to me. However, I believed that such a procedure with the quoted mortality at that time was too high and, therefore, with the mortality of 12 per cent in those days for end-to-end anastomoses, I tried the Rankin resection modification of the Mikulicz procedure because I thought that I could lower it. I have been able to lower it. I will continue to do it because in not a single case I did have I had a recurrence in the abdominal wall. What does that mean? It means that all the gland-bearing area is removed. The trouble with the old Mikulicz operation and even the Rankin resection operation is that the mesentery obviously is stuck up against the peritoneum of the abdominal wall when you do the operation. It is there when you close the colostomy. If you are going to have local recurrence in the mesentery, you are going to have local recurrence in the abdominal wall. I have not seen a single case of my own series which means to me that I can do a more radical operation with this procedure than I can with the end-to-end anastomosis in a good many segments because I do not have to worry about the blood supply. In the past, patients with peritonitis did not die from contamination following an open operation; they died from necrosis at the suture line. If I do not have to worry about that, I believe that I am going to have a lower mortality.

It seems to me in looking over all the evidence that the only controversy is about what level you are willing to do an anastomosis. Knowing what to do about malignancy, it does not make any difference.

Dr. Wangensteen started out a few years ago believing surgery could be done with lesions within 4 cm. of the anus. I think the last thing that he said was 8 or 10 cm. I believe that before very long he will say 12, 15 or 16 cm.

Those things were done in the old days at that level. For that reason it is nothing new.

What is new about it is anastomosing them below the reflection of the peritoneum and that is distinctly new. I do not have confidence in the operation. Of course, it can be done technically, but I believe that one must do an awful lot of them before you can do them technically. If you take the rank and file of all ages and all weights, it is an extremely difficult operation. It is a far more difficult operation than the abdominoperineal resection.

I came to do the radical operation and the Miles operation because I was brought up in a small way also. Readers are acquainted with the operations that were in vogue in 1915 to 1925. They were small operations. The Kraske operation, the pull-through operation; everything was done to try to save the sphincter. I would rather have a patient alive and well than have him have a local recurrence. As Miles often said, "The patient has been sitting on this cancer too long before he comes to me, and I am not going to contribute to him sitting on it any longer."

I am sure that the smaller operations are bound to lead to more local recurrences. As a matter of fact, there is a paradoxical figure here. David and Gilchrist, three months ago, showed figures in which their local recurrence was 20 per cent. Now, it is something new in malignancy and our conception of malignancy if we are going to have a lower local recurrence from a smaller operation. There is something wrong with the figures. Of course, I think, as Dr. Wangensteen has said, the time is too soon and the number of cases too small to draw final conclusions. It is an admirable attempt, however, to improve the situation and for that we give him credit. However, in the meantime one must use his own judgment as to what operation fits him best and the number of patients that he thinks he will be able to benefit.

I would say that if the percentage of recurrence, which I think is going to be higher with these smaller operations, is going to be higher than with the abdominoperineal operation, that alone is sufficient reason to do the abdominoperineal resection because there is little use in transplanting one morbidity with another? A man with cancer *in situ* in the rectum is ten times better off than one with a perineum which finally becomes necrotic from broken down malignancy.

In his paper Dr. Wangensteen reported that in certain lesions of the descending colon he may go up and remove the entire descending colon, the splenic flexure and so forth and anastomose the transverse colon to the rectum. He wrote that a man who does the abdominoperineal operation and the Mikulicz operation cannot stand on the same stage. I certainly cannot see how a man can stand on the same stage and be satisfied with getting 2 cm. below a low rectal growth and yet remove a foot or two of colon which is so absolutely and absurdly unnecessary because you never get glands up in the splenic flexure from a growth in the sigmoid or the descending colon. The vulnerable area, of course, is the gland, and there is just so much you can do; and if you tamper too much to get those glands out, you are going to have a very high mortality.

The mortality, as we know, has been brought down. This operation started in 1925 with Miles, and the mortality was 25 to 30 per cent. In this country it was lowered to 20, 15, 12, 10, 7, and so forth. That has been done by a combination of factors that I do not have to enumerate here. You know what they are: better preoperative treatment, better anesthesia, better care of the patient afterward, at least until the war emergency, all of those things. You cannot point to one factor which made the mortality less. Of course, I am small minded enough to believe that the only thing that has made a difference in my series is the use of steel wire sutures. Of course, everybody laughs at it, but that is just my fetish. I believe in my old experience and my new experience that it has immensely cut down mortality.

Today we have a series of 535 consecutive cases from 1942 to 1946 of abdominoperineal resection in one stage with twenty deaths, or a mortality of 3.7 per cent. Therefore, mortality is a thing of the past, I believe, with present-day good hands and good hospital facilities.

Ten of these deaths were from embolism, many of them on the day they were going home. You all know that story. But the one thing I would like to ask you is in connection with this embolism mortality and what is being advocated in Boston. Would you be willing to ligate 1,070 femoral veins in order to try to offset ten deaths from embolism? I do not know that I would at this time, because I am not absolutely sold on the idea of femoral ligation, to do it prophylactically as advertised in all malignant cases. I do not believe that I would

choose to do it in contemplating another series like this in the next five years.

Table I shows a group of cases from 1940 to 1944; 117 resections were done by the Mikulicz technic, with six deaths. When you do 117 of these cases in the sigmoid, you know what you

TABLE I
CARCINOMA SIGMOID OBSTRUCTIVE RESECTION

Year	Res.	Mort.	%
1940	16	0	
1941	21	3	
1942	23	1	
1943	22	0	
1944	35	2	
Total	117	6	5.1%

TABLE II
ABDOMINO-PERINEAL RESECTION

	Cases	Mort.	
1942	73	0	
1943	91	5	
1944	124	6	
1945	112	6	
1946	135	3	
Total	535	20	3.7%

are dealing with. Pieces of the bladder have been removed, the uterus may be removed, the adnexia have been removed, oftentimes a very large portion of the peritoneum is removed. Many of those cases are perforated at the time you get them, so that it seems to me the sigmoid is really by and large a bigger problem than the rectum for these complications. A mortality of 5 per cent in 117 cases is perfectly satisfactory to me because of the type of cases that they are done on and we have no local recurrences.

Table II demonstrates our group of 535 cases; there were 137 consecutive cases without a death. This was entirely too long, of course, and your luck cannot go that way all the time, as Dr. Wangensteen has pointed out. At any rate the series was a long one and we were interested in attacking it from all sides to see if we could find any one factor which was responsible.

I think we know a little bit more about cancer than we used to certainly; we do not know half enough. The glandular dissections and the information we have had from that is very important. However, I think the venous involvement is ten times more important than the glandular involvement.

In a group of 103 consecutive cases studied about ten years ago after the method of Duke and David and Coller, and so forth, we found 65 per cent of the glands involved. Then after reading some articles on pathology, particularly by Warren, we investigated this same group of cases again and without staining for elastic tissue, Dr. Graham was able to demonstrate venous involvement in 72 per cent of the cases, and he predicted that if he would stain all of these for elastic tissue to see where the malignant cells were, that he would probably do it in a higher percentage of cases. What does that mean? When you do a low anastomosis, you must tear through the mesentery fat. You may get 2 cm. below a good mucosa, but I believe the trouble is in the surrounding tissues, in the surrounding fat. Dr. Graham, I must say, laid more emphasis on invasion of the mesenteric fat as a prognostic thing than glandular involvement. I believe that it is true. The venous involvement is the important one. I believe unless you get very far around the growth, you are going to get local recurrence from these cells in the veins.

JOHN M. WAUGH (Rochester, Minn.): In attempting to cure malignancy by means of operation three important factors should be given consideration by the surgeon: First, does the operation he proposes to use carry the best chance of cure for the patient of any of the procedures available; second, is the risk of operation reasonable or does it outweigh the chances of cure; and third, is the patient left in a normal condition following surgery and able to carry on his work without impedient or embarrassment. Because in the past so many patients have of necessity had permanent and temporary colostomies following operations for cancer of the colon and rectum, it is fitting and timely that we periodically review our operative procedures to see if more patients cannot be spared colonic stomas without sacrificing a high curability rate or a low operative mortality.

In the past ten years we have seen the staged procedures for resection of the abdominal colon gradually give way to the single stage operation without temporary colostomy except in the

face of obstruction. Right hemicolectomy has been done in one stage at the Mayo Clinic for several years with immediate primary anastomosis with a mortality of 2 to 3 per cent. In 1946 eighty such resections were carried out without a hospital fatality. In the transverse colon and descending colon to the lower sigmoid resection with primary anastomosis has been done, as Dr. Wangensteen has shown, with a mortality of 5 per cent and less. In the past five years on my own service 197 resections with primary anastomosis have been done in the left colon with a hospital mortality of 5.5 per cent. This figure includes resections for carcinoma of the rectosigmoid, upper rectum and lower sigmoid. If one considers only the group of lesions that could have been exteriorized and excludes the lower group, there were 117 patients with a 4 per cent hospital mortality. I do not believe that this mortality could have been lowered by use of the exteriorization procedure. The length of hospital stay for the single stage procedure has averaged one-third that of the multiple stage. This is no small economic factor, especially if one elects to carry out palliative resections for obviously incurable disease.

Most of the controversy in recent years concerning rectal surgery has been (1) Is the sphincter conserving operation radical enough for cure whether it be done by anterior resection as advocated by Dixon and Wangensteen or by the pull-through procedure popularized by Babcock and Bacon? (2) Is the operative

TABLE 1

INCIDENCE OF RETROGRADE NODAL METASTASIS IN CARCINOMA OF RECTUM AND RECTOSIGMOID: REVIEW OF LITERATURE

	Total Cases	Positive Nodes (Cases)	Nodes Involved below Lesion (Cases)	Distance below Lesion	Site of Lesion
Westhues	74	?	1	1	?
McVay, 1922	100	47	1	0–1 cm.	Ampulla
Wood and Wilkie, 1933	100	51	0		
Gabriel, Dukes and Bussey, 1935	100	62	2	0–1 cm.	Upper rectum (8 and 9 cm.)
Gilchrist and David, 1938	25	16	2	−2 cm. −4 cm.	Ampulla
Coller, Kay and MacIntyre, 1940	33	22	1	1.5 cm.	Rectosigmoid
Grinnell, 1942	75	41	1	1 cm.	Ampulla (6 cm.)
Total	507	239(+)	8		
Glover, 1944	100	100	36		Upper rectum
			27	0–1 cm.	
			6	1–2 cm.	Rectosigmoid
			1	2–3 cm.	
			1	3–4 cm.	Lower sigmoid
			1	6–7 cm.	

mortality low enough and the functional result sufficient to justify these sphincter conserving operations?

First, as far as cure is concerned with these operations, I am satisfied personally that they are just as curative as the Miles procedure for

TABLE II
ANTERIOR RESECTION (NON-PALLIATIVE OPERATION): FIVE-YEAR SURVIVAL RATES FOR DIFFERENT LEVELS
(Dr. Dixon's Data)

Distance from Dentate Line, Cm.	All Cases		No nodal Involvement		Nodal Involvement	
	Number	5 Yr. Survival Rate, %	Cases	5 Yr. Survival Rate, %	Cases	5 Yr. Survival Rate, %
6–10	74	63.7	32	72.4	42	57.1
11–15	97	70.2	58	78.8	39	57.7
16–20	101	66.9	60	71.5	41	60.4
All Levels	272	67.7	150	74.0	122	58.5

lesions where the lower edge of the cancer is 5 cm. or more above the pectinate line. The work of Glover on retrograde spread (Table 1) predicted such would be the case and this has been substantiated by Dr. Dixon's five-year cure rate following anterior resection. (Table 11.) Second, the operative mortality of anterior resection is approaching that of the Miles operation but because of the suture line undoubtedly will always be a trifle higher even in the hands of those with considerable experience. The pull-through procedure, however, can be done with just as low a mortality as the Miles and in my own experience of approximately one hundred there has been one hospital death. Anal control after even low anterior resection has been excellent. Following the pull-through operation most patients do not have perfectly normal control and few are able to control flatus. The majority, however, rarely soil themselves unless diarrhea appears and all patients prefer the procedure, even those with the most unsatisfactory results, to an abdominal colostomy.

OWEN H. WANGENSTEEN (closing): All of us in a sense are surgical Fausts. I am not thinking of those who sell themselves for thirty pieces of silver or a mess of pottage. I am talking about compromises which surgeons are willing to make. Dr. Jones would have us believe that he affects less interest in preservation of fecal continence and sexual function than he does in the contour of the female breast. That is an interesting attitude of mind. Dr. Jones apparently finds no difficulty in defending the necessity of performing the abdominoperineal operation in all patients with cancer of the iliac colon and rectosigmoid in which the Mikulicz procedure cannot be done, an admission suggesting perhaps that he is a better master of the abdominoperineal operation than he is of primary resection. Certainly the time has come for experienced surgeons to exteriorize the exteriorization operation in dealing with cancer of the colon. In practiced hands, the two operations can be done at the same risk with an important salvage in hospital stay and expense to the patient for whom primary resection is done.

I grant freely that I have overextended myself in the anxiety to please a patient who wishes her sphincteric function saved. Like the young lady in the musical comedy "Oklahoma", I have found it difficult now and then to say no—but I am learning.

With reference to the recurrences which we have observed after the anastomotic operation, it perhaps is not out of place to emphasize that these have not been in the bowel wall primarily; on the contrary, the recurrences first made their appearance outside the bowel involving it secondarily. In other words, what we interpreted as a Dukes' group A case, on occasion belonged in reality to Dukes' group C, in that, invisible lateral spread already had occurred at the time of the initial operation but had not been identified as such. Our own experience suggests quite definitely that for the upper group (14 to 20 cm. from the anus) the anastomotic operation holds out just as much promise of cure as does the abdominoperineal operation. For patients with lesions less than 8 cm. from the anus we have given up the anastomotic procedure because of the greater hazard of local recurrence than follows the abdominoperineal operation. The controversial group, it seems to me, is the intermediate group between 9 and 13 cm. from the anus. There is no debate about the superiority of the abdominoperineal operation for this group; that is granted. The question is: How much better is it from the standpoint of curability of cancer than the anastotmotic pro-

cedure? This is the group in which some case selection may have to be done. Among thirty-two patients with lesions at this level for whom an anastomotic procedure was carried out, local recurrence was observed by us twice (6.3 per cent). With proper case selection I believe that the anastomotic procedure with satisfactory removal of the lymphatic drainage area can be done in a large number of patients with lesions at this level.

Dr. Waugh's figures are excellent. In fact, they are so good that one cannot help but conclude that some selection must have occurred in his groups.

With reference to the kind of anastomotic operation to be carried out our experience suggests definitely that the direct suture carried out as a one-stage procedure from the abdomen is the best operation. For anastomoses made at 8 cm. or more from the anus drainage is rarely necessary and a supplemental external decompressive vent is not necessary. With elimination of the anastomotic procedure for lesions lying less than 8 cm. from the anus the single row direct suture method becomes, save for a few exceptions, the standard procedure.

A young woman will put up with less complaint on this score, getting rid of an abdominal ileostomy, than will a young man. I know young marriagable girls who had an ileostomy for some time and they are closed. Even though they have four or five stools a day, they make no complaint about it. I suppose maybe the male is a more complaining creature. He will suggest two or three stools a day is a burden for him to bear.

Will you read the questions and I will try to answer them one by one, if I can.

Dr. Ault: Doctor, I understand this is in a research process. I am wondering how many anastomoses between ileum and involved rectum or rectosigmoid in cases of ulcerative colitis have held up for how long.

Dr. Wagensteen: Obviously, an attempt to conserve the rectum or part of it in the type of case demonstrated by Dr. Ault on the screen would be foolhardy. If one is going to anastomose the ileum to a residual rectal segment, that segment should be free of ulcerative colitis or have only punctate hemorrhages in the mucosa without evidence of destruction of the mucosa.

Dr. Ault: Thank you, Dr. Wangensteen. We want to make this clear to everybody. How many have had to be converted back to ileostomy?

Dr. Wangensteen: That is easy to answer —none.

Dr. Ault: What complications, stricture, abscesses and what morbidity have attended this procedure of anastomosing the ileum to involved rectum or rectosigmoid?

Dr. Wangansteen: There has been no mortality either accompanying operation or since operation. Whereas some of the patients with mild evidences of disease in the distal rectal segment have continued to have some diarrhea, it has been gratifying to observe that extirpation of 85 to 90 per cent of the extent of the disease has permitted the lesions in the residual rectal segment to improve. Moreover, migration of the disease into the ileum after such anastomoses has not been observed. Only one patient has been operated upon in a febrile state. To that patient we gave streptomycin, sulfasuxidine and penicillin prior to operation. A one-stage operation was done anastomosing the ileum to uninvolved rectum, excising the involved colon between. The patient convalesced uneventfully and left the hospital about a week after operation.

Some of the matters discussed today obviously are controversial in nature; however, we cannot resolve our differences by debate but rather by analyzing our experiences critically and recording them truthfully. That is what Dr. Toon and I have tried to do in this paper. Time is the final arbiter of all things. Until she has given her answer, let us be tolerant of our honest differences of opinion.

POSTERIOR LEVATOR SPACE ABSCESS*

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DEEP perirectal abscesses are of much concern to the surgeon. It is commonly believed that they occur within three deep perirectal spaces (right pelvirectal, left pelvirectal and retrorectal) and also within the wall of the rectum (intramural). On many occasions these abscesses remain unrecognized and progress for an indefinite period, thus permitting relatively simple abscesses to become complicated by spreading from one deep space to another, or by rupturing high into the rectum, causing that serious complication, the true "anal-rectal" fistula. The pathways by which infection spreads from the anorectum to these deep spaces never have been accurately understood or established on a strictly anatomic basis. My dissections of the pelvic diaphragm and its related structures reveal the presence of a fourth deep space, lying posterior and lateral to the anorectum and situated between the superior and inferior layer of the levator muscle. This space is apparently undescribed; to refer to it, I shall use the term, "posterior levator space."[1,2] In my clinical experience this space frequently is involved by a deep perirectal abscess.

RESEARCH MATERIAL

Pelves of eight human cadavers of the Negro and white races, male and female, were dissected in detail. Pelves of forty cadavers of the Negro and white races, male and female, were surveyed in the laboratory while dissected by others. This description is based upon a dissection made from both the perineal and pelvic approach and a comparison of the dissection with sagittal, parasagittal and coronal sections.

TERMINOLOGY

In clinical literature on this problem various terms have been applied to indicate certain useful anorectal landmarks. These have not always been identified on a strictly anatomic basis by their originators; hence, others using these terms have frequently introduced confusion into the field. In some instances the inventors of the terms may have been confused themselves. For clarification, I shall define and limit the terms to those in use at the Philadelphia proctologic clinics, indicating the specific anatomic entities intended.

True Anal-Rectal Fistula. This is a very rare type of fistula; one in which the primary opening occurs in one of the crypts along the anorectal line and the secondary opening, high in the rectum.

Anorectal. This term is an adjective used to refer to the general region of the anus and the rectum. When referring to an anorectal fistula, it means a fistula in which the primary (internal) opening is either in the anus, at the anorectal line or in the rectum. It does not mean that the primary opening is in one and the secondary opening is in the other.

Anorectum. This noun refers to the general region of the anus and the rectum.

Combined Longitudinal Muscle Layer of the Rectum. This phrase describes that thickened portion of the longitudinal muscle layer of the rectum which extends

* From the Anatomical Laboratories of the Graduate School of Medicine of the University of Pennsylvania; under the direction of Oscar V. Batson, M.D., Professor of Anatomy. The anatomy, as it appears in this paper, was presented by the author in a paper, read before the American Proctologic Society, at the annual meeting, San Francisco, California, June, 1946. This paper received the Hermance Award, given by the American Proctologic Society.

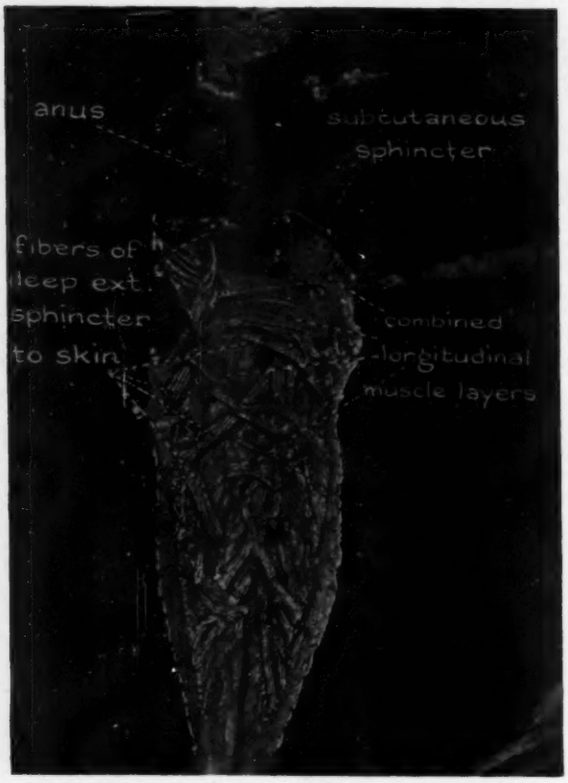


Fig. 1.* Retouched photograph, showing the insertions of the subcutaneous sphincter, the deep sphincter and fibers from the combined longitudinal muscle layer of the rectum, into the skin along the posterior midline. An incision has been made through the skin in the midline (anococcygeal skin sulcus) and extended into the anal canal. The skin edges have been pulled apart and held by pins. (Dissection by the author.)

inferiorly from the level of the levator fascia to the perianal skin below. It is composed of the longitudinal muscle layer of the rectum, fibers from the levator fascia and muscle fibers from both the superior and inferior layers of all three portions of the levator muscle (puborectalis, pubococcygeus and iliococcygeus).

Levator Fascia. This is the fascia covering the superior surface of the levator ani muscle.

Iliorectococcygeus Muscle. This is a new anatomic term proposed for the rectococcygeus muscle because this muscle is formed mainly by fibers from the iliococcygeus (striped), fibers from the longitudinal muscle layer of the rectum (smooth) and a few fibers from the levator fascia.

Circumanal Space. This is another new term proposed for a space which encircles the anus. It lies between the inferior edge of the internal sphincter muscle and the anal intermuscular septum. Its inferior boundary is formed by those fibers from the combined longitudinal muscle layer which form the anal intermuscular septum and insert into the skin of the anal canal. Some of these fibers also pass upward, medial to the internal sphincter, to insert into the anal skin at a higher level, thereby forming a "U-shaped" sling around the inferior edge of the internal sphincter.

Parietal Layer of the Pelvic Fascia. This refers to that portion of the pelvic fascia surrounding the muscles, blood vessels, nerves and lymphatics of the pelvis.

Coccygeal Muscular Raphe. This is a proposed new term to indicate more accurately the musculo-fibro-tendinous insertions of the inferior layer of the levator (puborectalis, pubococcygeus and iliococcygeus) into the tip and sides of the coccyx; formerly referred to as the "anococcygeal raphe." The term "anococcygeal raphe" is a misnomer since this web of muscle fibers does not attach to or come in contact with the anus in any place.

Anal Fascia. This term refers to the fascia covering the inferior surface of the levator ani muscle and the external sphincter muscles.

ANATOMY

As the individual muscle bundles of the levator pass medially (Figs. II-1 and IV-4), they divide near the rectum into a superior (Figs. II-2 and IV-2) and an inferior (Figs. II-3 and IV-3) layer. These two layers diverge from each other slightly in a wedge-shaped manner (like a letter "V" lying on its side, with the open end toward the midline), thereby forming an anatomic space which is lateral and posterior to the

* Due to the fact that the relationships of a single structure may be mentioned in several different locations, Arabic numerals have been used in the text to indicate each specific relationship in its various locations. Thus (III-9) as it appears in the text, refers to the "retrorectal space" (9) on Figure III.

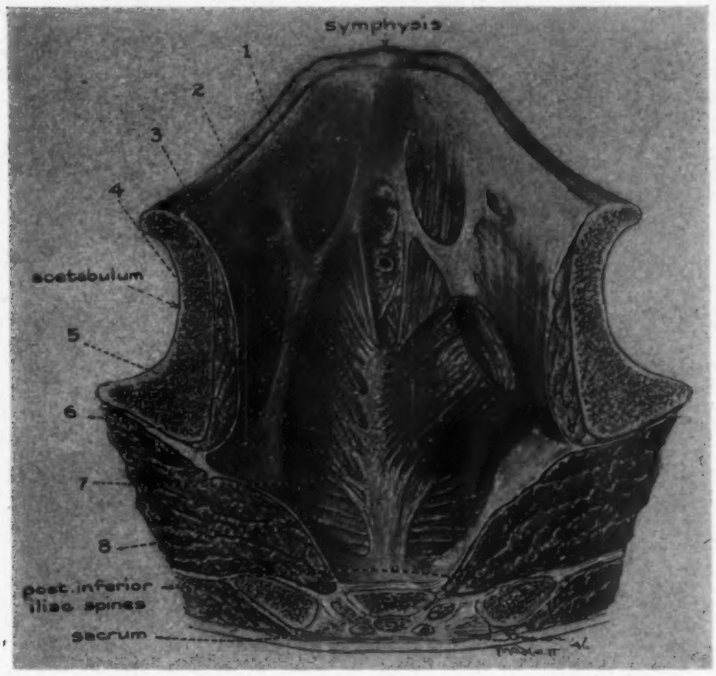
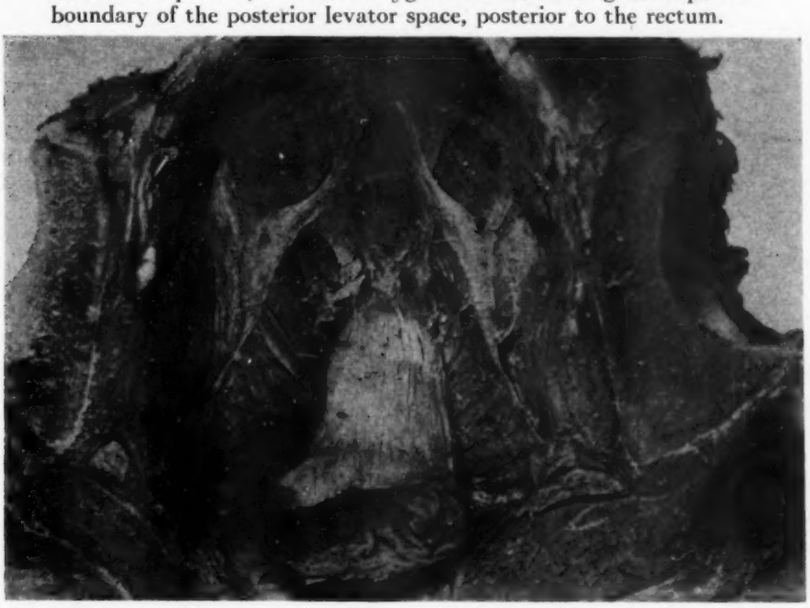


FIG. 11. Drawing showing superior view into dissected male pelvis, the rectum being displaced anteriorly and to the right. (Dissection by the author.) 1, Levator muscle lateral to the point of division into a superior and an inferior layer. 2, Superior layer of the levator. 3, Inferior layer of the levator. 4, The row of fossules with connecting tracts which extend inferiorly to the circumanal space (level of anal-intermuscular septum). 5, Superior layer of levator forming the superior boundary of the posterior levator space to the side of the rectum. 6, Longitudinal muscle layer of the rectum giving off fibers posteriorly, which unite with fibers from the iliococcygeus and fibers from the levator fascia, to form the iliorectococcygeus muscle. 7, The lateral margin of the iliorectococcygeus muscle, the usual point of "break through" for abscesses from the posterior levator to the retrorectal space. 8, Iliorectococcygeus muscle forming the superior boundary of the posterior levator space, posterior to the rectum.



A

FIG. 11. A, Actual photograph showing superior view into dissected male pelvis; as shown in Figure 11. (Dissection by the author.) Only structures on the left side of the pelvis have been dissected.

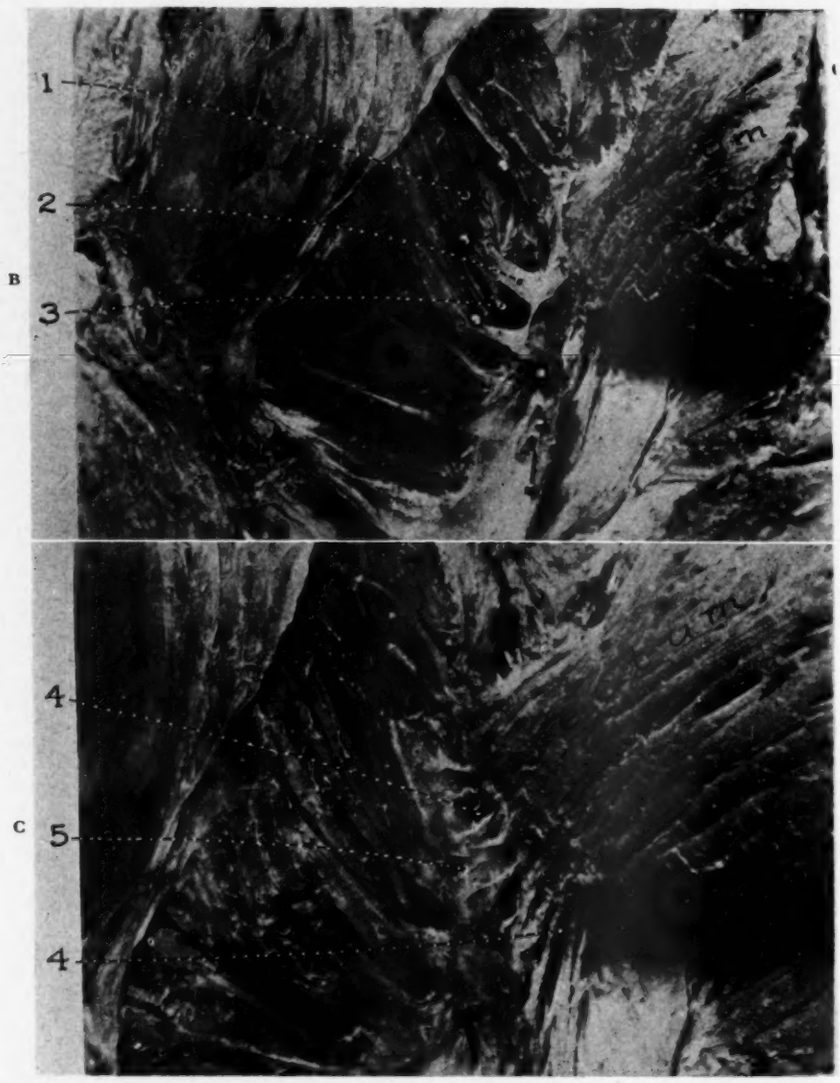


FIG. 11. B, actual close-up photograph showing the manner in which the individual muscle bundles of the levator divide into a superior and an inferior layer. The left edge of the iliorectococcygeus muscle is lifted up by pins. (Dissection by the author.) 1, Individual muscle bundle of the levator muscle (before division). 2, Superior layer of the levator (individual muscle bundle). 3, Inferior layer of the levator (individual muscle bundle). C, showing the row of fossules, lateral and posterior to the rectum. (Dissection by the author.) 4, Row of fossules with connecting tracts which extend inferiorly to the circumanal space. 5, Superior layer of levator forming the superior boundary of the posterior levator space to the side of the rectum.

rectum. This space is against the rectal wall; its internal boundary is the combined longitudinal muscle layer of the rectum (Fig. III-1 and 5) in the male and the layers of the rectum and vagina in the female.

The superior layer of the levator unites with fibers from the longitudinal muscle layer of the rectum (Fig. II-6) and the fibers of the levator fascia to form the iliorectococcygeus muscle. (Figs. II-8, III-8 and IV-1.) The "posterior levator space," (Fig. III-3) lies beneath the iliorectococcygeus muscle, surrounding the rectum posteriorly and laterally, in the form of a horseshoe.

As the superior (Figs. III-6 and IV-5) and inferior (Fig. IV-6) layers of the levator come in contact with the rectal wall, each

D

FIG. II. D, actual close-up photograph showing the manner in which the individual longitudinal muscle fibers of the rectum pass into the iliorectococcygeus muscle. (Dissection by the author.) 6, Fibers from the longitudinal muscle layer of the rectum passing posteriorly into the iliorectococcygeus muscle. 7, The usual point of "break through" for abscesses from the posterior levator to the retrorectal space. 8, Iliorectococcygeus muscle.

layer gives off thin bundles of muscle fibers to the combined longitudinal muscle layer of the rectum. In doing so, they form a series or row of fossules (Fig. II-4) with connecting tracts which extend inferiorly to the circumanal space (Fig. IV-7) [the level of the anal intermuscular septum (Figs. III-15 and IV-8)]. Infection spreads from the anal ducts to these tracts, and thence upward to the various deep perirectal spaces. These tracts lie within the combined longitudinal muscle layer of the rectum. Behind the rectum the tracts to the "posterior levator space" lie posterior to the corresponding ones to the retrorectal space.

Heretofore, we have recognized and defined three deep perirectal spaces, all lying above the parietal layer of the pelvic fascia: (1) the retrorectal (Fig. III-9) space, (2) and (3) the right and left pelvirectal spaces. This paper deals with the discussion of a fourth deep space, the "posterior levator space." (Fig. III-3.)

The superior boundary of this space is formed posterior to the rectum by the iliorectococcygeus muscle (Figs. II-8, III-8 and IV-1) and to the side by the superior layer of the levator. (Figs. III-4 and II-5.) The inferior boundary of this space is formed posterior to the rectum by the coccygeal muscular raphe (Figs. III-7 and IV-11) and to the side by the inferior layer of the puborectalis muscle. (Figs. III-2 and IV-10.)

The deepest part of the "posterior levator space," that is from the top to the bottom, lies posterior and posterolateral to the rectum. As the space approaches the pubis it gradually decreases in depth and width. This space is filled in by a loose layer of fatty, areolar tissue which is readily separated with the blunt probe. Hence, an abscess of the "posterior levator space," as shown by the arrow (Fig. III-3), may spread around the bowel from back to front, and from one side of the pelvis to the other by contiguity.

Due to the fact that this fourth deep space is entirely bounded by subdivisions of the levator muscle and because the most extensive part of this space lies posterior to the rectum, I have given it the descriptive anatomic name, "posterior levator space." (Fig. III-3.)

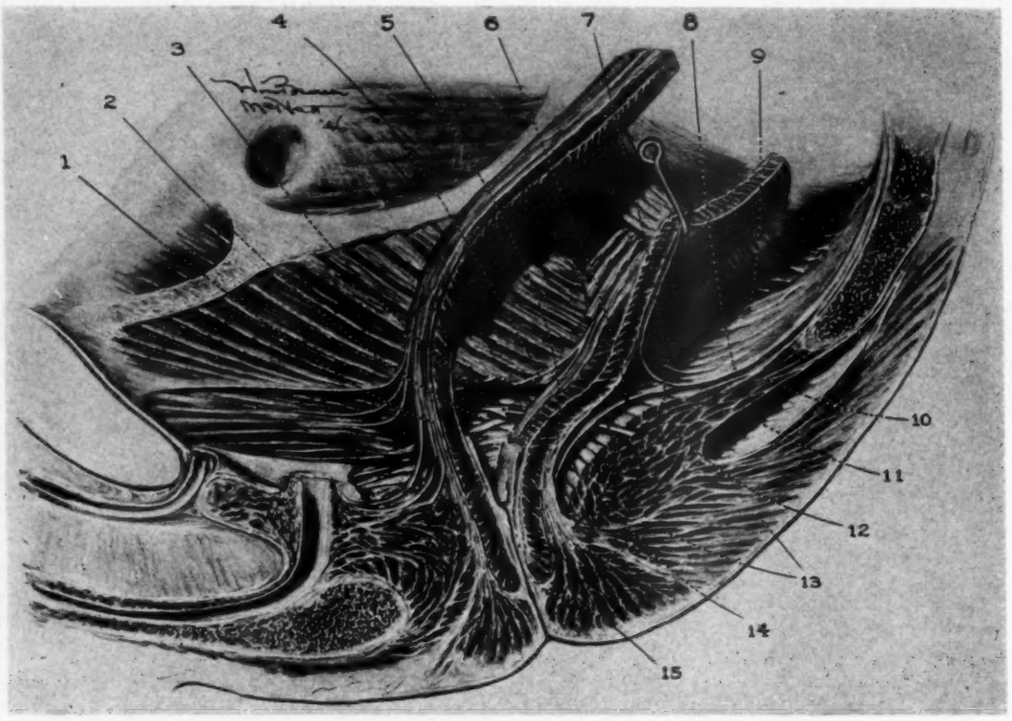


FIG. 111. Drawing of mid-sagittal section somewhat schematic, showing the relationships of the anorectum to the pelvic diaphragm and the anorectal musculature. A window has been cut through the lateral wall of the rectum, layer by layer, to show the relationships lateral to the rectum. (Dissection by the author.) 1, Combined longitudinal muscle layer (anterior to the rectum). This layer is composed of the longitudinal muscle layer of the rectum, fibers from the levator fascia and fibers from both the superior and inferior layers of the levator. 2, Inferior layer of the puborectalis muscle. 3, Arrow lying in the posterior levator space. This space surrounds the rectum like a horseshoe, with the open end of the horseshoe toward the pubis. 4, Superior layer of the levator muscle. 5, Combined longitudinal muscle layer of the rectum (posterior to the rectum). 6, Fiber from the superior layer of the levator, to the combined longitudinal muscle layer of the rectum. 7, Inferior boundary of the posterior levator space formed behind the rectum by the superior surface of the coccygeal muscular raphe. 8, Iliorectococcygeus muscle formed by fibers from the iliococcygeus (striped), fibers from the longitudinal muscle layer of the rectum (smooth) and a few fibers from the levator fascia. This muscle forms the superior boundary of the posterior levator space behind the rectum. 9, Retrorectal space. 10, Coccygeal muscular raphe attaching to the tip and sides of the coccyx. 11, Posterior subsphincteric space (connecting the two ischioanal fossas). 12, Puborectalis muscle (the sling of the puborectalis behind the rectum). 13, Fibers of the deep external anal sphincter muscle inserting into the skin along the anococcygeal skin sulcus. 14, Deep external anal sphincter muscle (posterior to the rectum). 15, Anal intermuscular septum.

CLINICAL MANIFESTATIONS

In all patients observed to date infection began in one of the posterior crypts and extended along the tracts mentioned above to the "posterior levator space."

Infection may be confined to the "posterior levator space" alone but more often there are concurrent abscesses of the retrorectal space or one of the pelvirectal spaces. In some subjects all of the perianorectal spaces, both superficial and deep, were involved. Multiple tracts, emanating from a single crypt, may extend to form simultaneous abscesses in the "posterior levator" and retrorectal spaces.

The extension from the "posterior levator space" to the retrorectal space usually takes place at the lateral margin of the iliorectococcygeus muscle (Fig. 11-7) although this occasionally occurs posteriorly in the midline. Lateral extension between the retrorectal and the pelvirectal space is common, whereas a lateral upward extension from the posterior levator to the overlying pelvirectal space is less common.

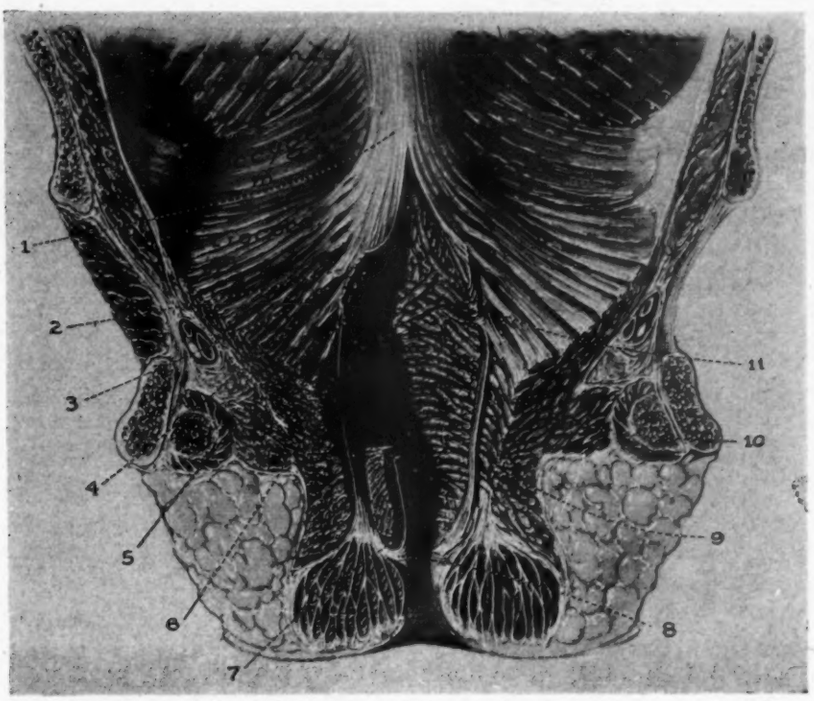


FIG. IV. Drawing of coronal section of dissected male pelvis, somewhat schematic; showing the extent of the posterior levator space posterior to the rectum, and the manner in which the inferior layer of the levator winds itself around the rectum to form the deep external sphincter muscle of the anus. The iliorectococcygeus muscle has been split in the posterior midline and the flaps turned upward. The wall of the posterior half of the rectum has been removed, with the exception of a few muscle bundles of the combined longitudinal muscle layer on each side, and a block of the lower end of the internal sphincter muscle on the right. The skin of the anal canal has been removed. (Dissection by the author.) 1, Iliorectococcygeus muscle. (Superior boundary of the posterior levator space, posterior to the rectum.) 2, Superior layer of the levator. 3, Inferior layer of the levator. 4, Levator muscle (lateral to the point of division into a superior and an inferior layer). 5, Muscle bundle from the superior layer of the levator to the combined longitudinal muscle layer of the rectum. 6, Muscle bundle from the inferior layer of the levator to the combined longitudinal muscle layer of the rectum. 7, Circumanal space. 8, Anal intermuscular septum. 9, Deep external anal sphincter muscle. 10, Puborectalis muscle; inferior layer as it winds and entwines itself around the bowel to form the deep external anal sphincter muscle. 11, Coccygeal muscular raphe (inferior boundary of the posterior levator space behind the rectum).

SYMPTOMS AND DIAGNOSIS

The symptoms of "posterior levator space" abscess are the same as those of any deep perirectal abscess. However, there are several points which help one to make a diagnosis of "posterior levator space" abscess. External evidence of the suspected infection is usually absent, but digital pressure between the tip of the coccyx and the anus produces extreme deep pain. The circumscribed bulge of the abscess into the rectum is readily palpated in the posterior midline. Bidigital examination often reveals the abscess to be under marked tension and protruding into the rectum like a large walnut. In some patients fluctuation is present. Occasionally, the abscess tends to rupture into the rectum laterally, with the bulge occurring in this position. *Due to these facts, abscesses of the "posterior levator space" have been probably mistaken for intramural or retrorectal abscesses in the past.*

SURGERY

In considering surgery several points should be emphasized:

1. Complicated "posterior levator space" abscesses with their inevitable fistulas are often best operated upon in stages. The first stage should include incision and drainage, with location of the primary crypt and tract through which the infection originated. If it is not advisable to remove the primary tract at this time, a seton is placed through the tract, to be removed later.

2. These deep abscesses are opened under direct vision, layer by layer.

3. "Posterior levator space" abscesses are best opened and explored through a posterior midline incision; this separates rather than severs the fibers of the external sphincter and the coccygeal muscular raphe.

4. For counter drainage of the lateral extension of the "posterior levator space," a curvilinear incision is made lateral to the external sphincter muscle. This incision is carried anteriorly to the full extent of the abscess cavity and posteriorly to the lateral margin of the posterior insertion of the external sphincter (which should not be cut). After passing through the ischioanal fossa, the inferior layer of the levator muscle, which is covered by the anal fascia, is identified and incised, thus opening the lateral extension of the "posterior levator space." The skin edges are trimmed to prevent inversion. One or more Penrose drains are passed into the primary incision and out through the counter drainage incision and fastened.

5. "Posterior levator space" abscesses should never be drained through the rectum, as a "true anal-rectal" fistula may result.

CONCLUSIONS

Operations designed to cure "posterior levator space" abscesses should be definitely considered under the category of major surgery. Since an accurate knowledge of the pelvic diaphragm is of major importance to the success of these operations, this detailed description of a fourth, deep perirectal space, heretofore undescribed, should prove invaluable in anorectal surgery.

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2. Courtney, Harold. The posterior levator space (its relation to postanal infection). *Tr. Am. Proct. Soc.*, 45: 450–455, 1946.

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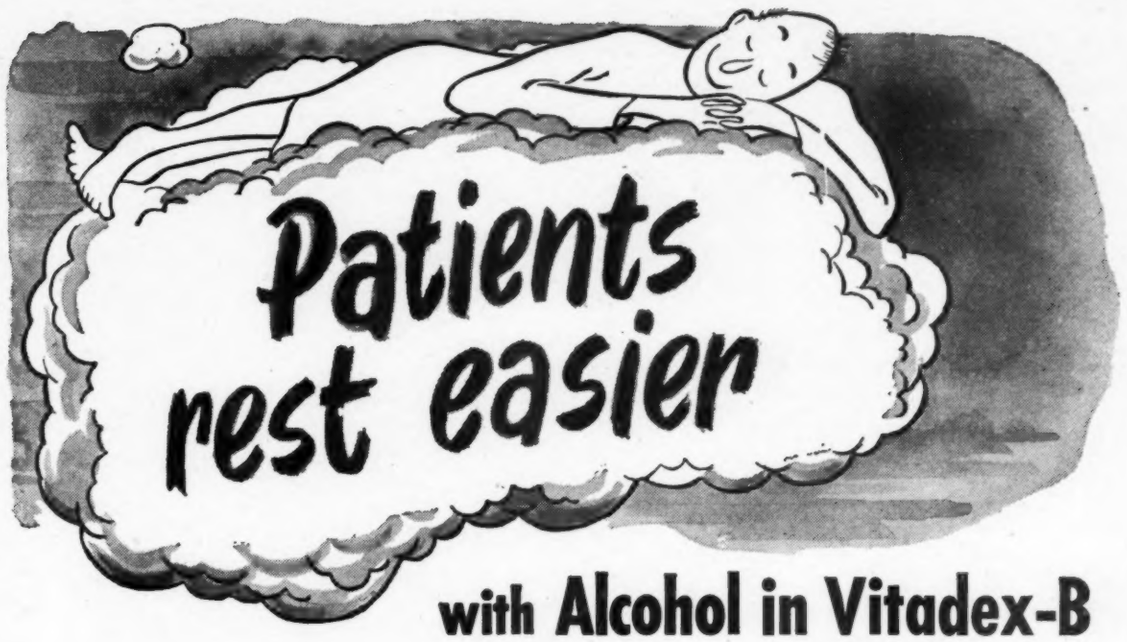

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Moore, D.C. and Karp, M., Surg. Gyn. Obst., 80:523-525, May, 1945
Craddock, F H., Jr., Craddock, F. H., Sr., Mr. of Med. Assoc. of Alabama, Nov., 1942

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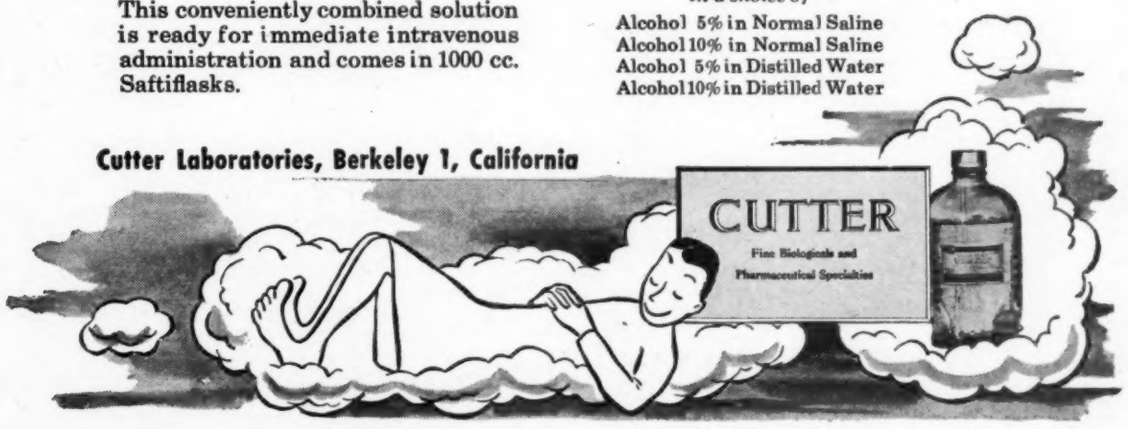

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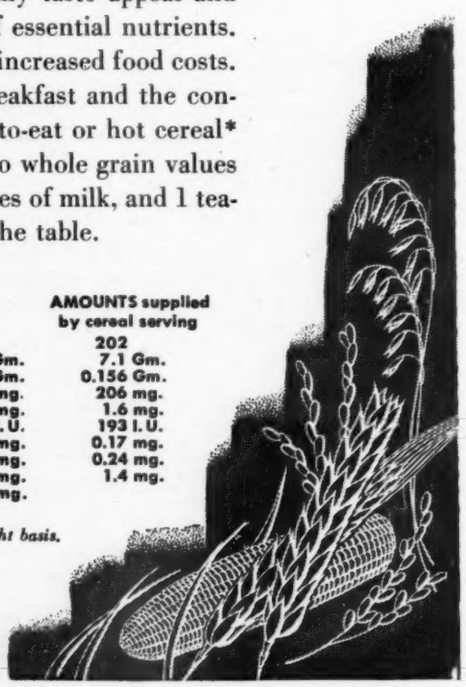

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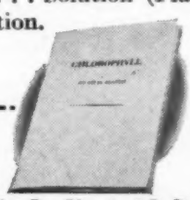

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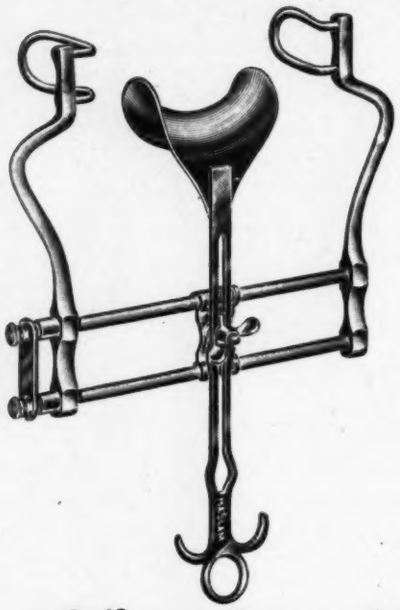

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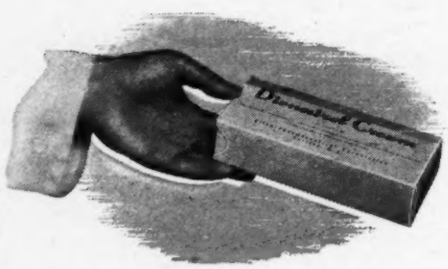

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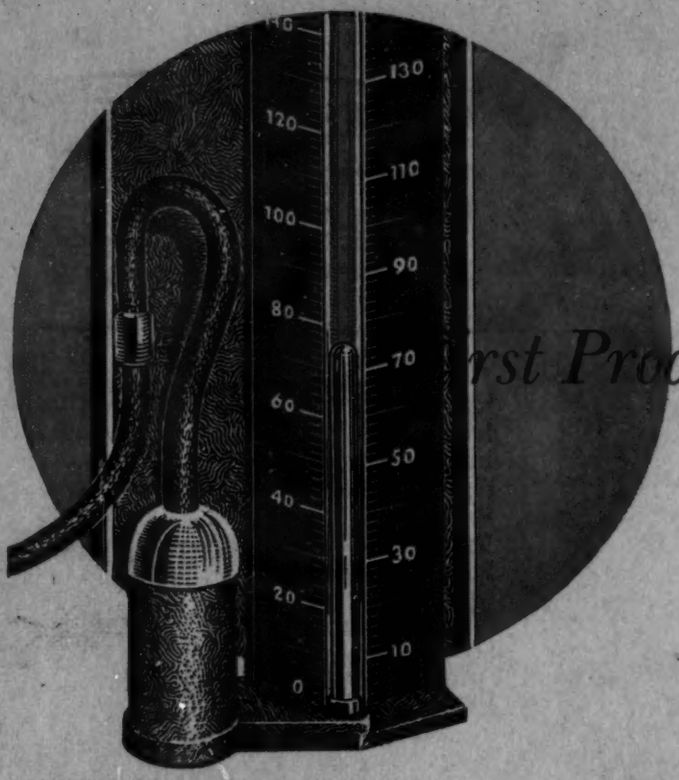

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*Gunther, Engelberg, and Strauss: Am. Jl. of Med. Sci. 204: 271, 1942

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